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CONTENTS OF PREVIOUS NUMBERS

SEPTEMBER, 1921. NUMBER 3

The Neurologic Dilemma. Sidney I. Schwab, M.D., St. Louis.

Intracranial Telangiectasis: Report of Two Cases. E. M. Hammes, M.D., St. Paul.

The Contrast Between the Brain Lesions Produced by Lead and Other Inorganic Poisons and Those Caused by Epidemic Encephalitis. G. B. Hassin, M.D., Chicago.

Studies in Asymptomatic Neurosyphilis: I. A. Tentative Classification of Early Asymptomatic Neurosyphilis. Albert Keidel, M.D., and Joseph Earle Moore, M.D., Baltimore.

A Comparative Study of the Sugar Content of the Spinal Fluid in Diseases of the Nervous System. Lewis D. Stevenson, M.D., St. Louis.

Epidemic Encephalitis (Lethargic Encephalitis): Its Psychotic Manifestations with a Report of Twenty-Three Cases. Leslie B. Hohman, M.D., Baltimore.

Abstracts from Current Literature:
Nissl's Staebchenzellen in the Cerebral Cortex of General Paralysis, Etc.—Epidemic Encephalitis.—Speech, Its Development and Integration with the Intelligence.—Sensory Aphasia, with Remarks on the Pathology of Memory.—Clinical Endocrinology.—Mendelian Law as Applied to the Psychoses.—Anemia in Old Age.—Psychopathic Child.—Spinal Cord Symptoms in Pernicious Anemia.—Compensatory Mechanism of Delusions and Hallucinations.—Variations in Intensity of Affective Reactions.—Sacral Sympathetic Trunk of the Cat.—Tertiary Syphilitic Psychoses Other Than Paresis.—Nerve and Plasmoderma.—Undiagnosed Cases.—Taste-Buds of Elasmobranches.—Thoracic White Ramus Communicans in Man.—Melancholia.—Feeblemindedness in Neurosyphilis.—Cerebral Softening.—Paralysis of Left Recurrent Laryngeal Nerve.—Colloidal Gold Curve in Epidemic Encephalitis.—Spinal Fluid in Epidemic Encephalitis.—Discrete Thermal Changes in Spinal Syphilis.—Weichbrodt's Sublimate Reaction in the Cerebrospinal Fluid.—Von Recklinghausen's Disease in the Negro.—Disappointments of Endocrinology.

Book Reviews.

OCTOBER, 1921. NUMBER 4

Calcification of the Cerebral Vessels with a Clinical Picture Simulating Brain Tumor. Peter Bassoe, M.D., and George B. Hassin, M.D., Chicago.

The Intraneural Plexus of Fasciculi and Fibers in the Sciatic Nerve. John Charnley McKinley, M.D., Minneapolis.

Hemihypertrophy and Mental Defect. Arnold Gesell, M.D., New Haven, Conn.

Eye Signs in Intracranial Tumors of the Anterior Fossa with Report of Two Cases. Albert B. Siewers, M.D., Syracuse, N. Y.

Results in Two Hundred and Fifty-One Cases Five Years After Admission to a Hospital for Mental Diseases. Earl D. Bond, M.D., Philadelphia.

Abstracts from Current Literature:
Grasp Reflex with Brain Tumor.—Changes in the Brain in Lethargic Encephalitis.—Personal Results in the Intraspinal Treatment of Neurosyphilis.—Pathogenesis of Catatonic Stupor.—Simultaneous Multiple Cloni in Epidemic Encephalomyelitis.—A Study of the Spinal Fluid in One Thousand Eight Hundred and Sixty-Nine Cases of Syphilis in All Stages.—Some Practical Points in the Organization of Treatment of Syphilis in a State Hospital.—Differential Diagnosis Between Myoclonic Uremia and Epidemic Encephalitis.—Abnormal Localization of Tetany in the Adult.—Brain and Thyroid Function.—Regarding a New Superficial Reflex—the Palmo-Mental Reflex.—Substantia Nigra.—Placental Transmission of Epidemic Encephalitis.—Isolated Neuritis of the Motor Branch of the Trigeminal Nerve.—The Mental Clinic and the Community.—Clinical and Anatomopathologic Contribution to the Study of Heredity Ataxia.—New Researches on the Morphology and Behavior of the Constituents of the Myelin Sheath in Secondary Degenerations.

Society Transactions:
Chicago Neurological Society.

Book Reviews.

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A CASE OF SLEEP LASTING FIVE YEARS, WITH LOSS OF SENSE OF REALITY*

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At a meeting of the Société de Neurologie de Paris, held the beginning of May, I was delegated to present the cordial greetings of that society to the American Neurological Association. I assure you that it affords me much pleasure to be the bearer of such greetings and to have the opportunity of expressing to you my appreciation of the hearty welcome that you have given me. I also wish to thank you for the interest that you have shown in my studies in pathologic psychology. Disorders of the psyche play an important rôle in nervous diseases and to understand such disorders often is essential. Hence I am led to present to you this study of the remarkable case of a young woman, aged 23, whom I was able to observe for several years at the Salpêtrière: a case of abnormal sleep prolonged for more than five years and of that strange disorder of apperception which seems to rob things of their reality, a disorder which presents many interesting problems.

HISTORY OF THE CASE

There is little to be said of the heredity of the patient. A grandmother, whom the patient resembles, was nervous and easily agitated. However, this young woman had always been quite different from her brothers and sisters and had been regarded as markedly neuropathic. She was unstable; sometimes very gay and exalted, at other times seclusive and gloomy. Complaining continually of extreme weariness, which came on rapidly, she worked very irregularly, although possessed of marked ability and rather unusual intelligence. Menstruation began at the age of 16, and has always been very irregular. The occupation she was to learn fatigued and distressed her, and at about the age of 13 she began to have paroxysmal disorders of consciousness in the form of fainting spells more or less prolonged.

* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

The first bad attack occurred at the age of 14 during a religious ceremony and was explained by the patient as follows: "This feeling always takes me when I am placed in a difficult situation—when the illumination is too brilliant or there is too big a crowd; or when I must do anything that is fatiguing or enervating. In a few minutes I am gone; I do not know what I am doing; I act like a somnambulist, an automaton; I do things without knowing that I do them, and no doubt I do stupid things. It is not I who order my movements; it is no longer I who act and speak." When the attacks were especially severe, she would gradually become motionless in a chair, close her eyes and apparently fall asleep. One would have to undress her and put her to bed, where for several hours she would remain inert. As time went on, attacks of this sort grew more and more frequent; they also were of longer duration, and at the age of 17 she began to sleep continuously.

CONDITION OF PATIENT ON ADMISSION TO HOSPITAL

She was admitted to the Salpêtrière, where for the last four years I have been observing her under favorable conditions. When brought to the hospital the patient was apparently sound asleep, and she remained in this condition almost without change for the first year. With eyes closed she scarcely stirred in bed, made no reply to questions and reacted but slightly if handled or pinched. The noise made by other patients did not seem to interest her in the slightest. During the bombardments of the war she gave no evidence of being disturbed, even when a shell burst near by. She asked for nothing, and did not open her mouth when offered food. However, she was easily fed without a tube. When the nostrils were pinched shut she opened her mouth a trifle and food placed in her mouth was promptly swallowed. She was badly constipated, there being no movement except by enema every second day. The urine was quietly passed in bed. Menstruation occurred only every three or four months. From time to time, especially during menstruation, sleep was disturbed by convulsive attacks, with large movements, during which the patient was almost always thrown out of bed. During such spells the patient spoke not a word and showed no signs of delirium. The convulsion lasted a few minutes, after which she slept as calmly as before.

Under these conditions one might easily believe that psychologic processes were reduced to the minimum—which would be a serious error. The parents of the patient had already informed me that it was well to leave at her bedside cakes, fruit and other food. Sometimes, during the night, when she thought she was not observed, she would stretch out her hand, take some of the food and eat a part of it.

EXPERIMENTS IN AROUSING THE PATIENT

I soon found that if I persistently spoke to her I could elicit a faint smile, accompanied by slight movements of the hand. I then began to talk to her just as if she could understand all I said. I explained to her that I wished to mesmerize her in order to get her into a different somnambulant state in which she would be able to reply to my questions. This conversation, which was accompanied by mesmeric passes, or simulated passes, gradually brought about a change in the patient's attitude. Little by little she was brought to open her eyes, sit up in bed and to talk with me in the most intelligent way. I thus secured about once a week a talk of from a quarter to a half hour, after which time the patient seemed fatigued, replied with greater effort and then dismissed me with an odd expression, to be explained presently: "Why do you want me to talk to you? You do not exist, neither do I. Good night." And she would lie back on the bed and fall into slumber lasting a week.

During these supposed hypnotic séances and the short conversations that I was thus able to have with her, I succeeded in getting her to promise that from time to time, when she could, in the same manner as she took the cakes during the night, she would take the pencil and paper that I placed within her reach and write me a letter. This "game" seemed to please her, as before long she got into the habit of rousing on certain nights when she thought no one was looking and of composing for me long rambling notes, which she then carefully concealed in a hole in the mattress lest they fall into the hands of any one but myself. Things went on thus during the first two years. Her prolonged sleep was interrupted by these short conversations about once a week, and two or three times a month she would write me a letter. Although these interruptions were very short, they gave me an insight into the peculiar mental state which accompanied her prolonged slumber.

PHYSICAL AND MENTAL CONDITION

First, it is to be noted that motion, sensation and elementary psychic processes were entirely intact, except that possibly during the convulsive seizures there was some blunting of sensation and less force of the movements on the right side. The reflexes, which were repeatedly examined and further investigated by Dr. Souques, were entirely normal. From time to time, cramps or contractions of the abdominal muscles were observed, but were easily dispelled by a little massage. There was some retraction of the right foot, apparently caused by the weight of the bed clothes, extending over a long period of time, and antedating her admission to the hospital. In short, there were no signs of organic disease of the nervous system. Evidently we had to do with a psychopathic disorder.

In investigating the mental state of the patient we must not be led astray by the first impressions she tried to convey by her conversation and letters. She insisted that she was in a state of frightful delirium—of profound dementia: "I spend all my time," she said, "in a horrible state of wandering. Pay no attention to a single word I say. I don't know where I am, I understand nothing of what is going on, remember absolutely nothing. The crazy people around me are far more sensible than I. At least they know what they are dreaming about. I am lower than the worst insanity. I am a living corpse. For me all is unconsciousness and irrationality in the midst of darkness and nothingness." This exaggeration and this pathos put us on our guard; we easily recognized "ce jeu des combles" of the hyperconscientious or phobic patient.[1] It was easy to see that she had neither the dementia nor the delirium which she imagined. On the contrary, she exercised her intellectual faculties in a most interesting way.

In spite of her assertions to the contrary, she knew perfectly where she was and repeatedly made very apt and amusing comments on members of the personnel and on the other patients, whom she called her worshippers because they came to gaze with wonder at the persistent sleeper. She laughingly told about another patient taking her hand and telling her fortune from the lines of the palm. She complained of a nurse coming at night and taking chocolates from the bag that her relatives had left on the stand by the bedside. One day she even requested me to ask her parents to send her some articles for a layette as she had noticed that one of the nurses was pregnant. The same keenness applied to recollections of her early life. In her letters she recounted amusing details from every period of her life.

Although she had received only an elementary education, she showed remarkable intelligence and a lively imagination. In the papers that she wrote for me were all sorts of philosophic ideas and endless discussions of metaphysical problems, which seemed intensely to interest her. As she said herself, these speculations attracted and at the same time were unwholesome for her. It was often after meditations of this sort that formerly she had had the disorders of consciousness and fainting spells. She also wrote little romances, puerile in conception but rich in imagination. She showed a peculiar taste for poetry and gave me many verses which she said she had written. Persons to whom I showed them thought they recognized something familiar, and very likely this was true. Probably she more or less consciously mixed memories with her own compositions. None the less, one received the impression that here was an unusually active intelligence, which appeared all the more strange in view of her prolonged sleep and apparent incontinence. When reminded that in

1. Obsessions et psychastenie **1**:61, 63, 1903.

writing all these things she couldn't be so stupid, she angrily replied: "One may be an idiot for real life but intellectually not one at all. Don't you know that one may be a fool in some things and not in others?"

THE SENSE OF UNREALITY

It is, indeed, in what she called real life, or rather in the sense of the reality of life, that we shall find very interesting psychologic disturbances. First, let us mention the feeling of automatism; "For a long time," she averred, "I have been acting only by reason of some incomprehensible instinct without knowing what I do . . . My will counts for nought . . . The person who makes me act must be outside of me. I speak without willing it, and if I don't talk foolishness it's no fault of mine. . . . With astonishment I hear myself talking," . . . etc.

The most interesting sensation is the feeling of unreality in all its forms: "I don't see things as I used to. . . . I see things devoid of substance. . . . They must be hallucinations and not real objects. . . . Things act only on my eyes, not on my brain. Doubtless I see everything, without doubt nothing is changed, except that things are not real. . . . It reminds me of the time when I had to take castor oil and some one would pinch my nose so I should not get the odor. I perceive things, but I have no sense of their taste." She described numerous occasions on which the feeling of unreality took possession of her: "When I did something too quickly, when I ran anywhere, I would suddenly stop, quite astonished, for my indolent brain had not been as quick as my legs; it had remained at the starting point and I was surprised to find that I had arrived. I had fallen from the moon and things no longer existed as they had. True, the appearance of things is just the same. I could describe them to you just as you would yourself, but what I see is only a play, a Punch and Judy show; it is clumsy, vulgar, unpleasant and, above all, false; it doesn't really exist."

She not only had doubts as to the existence of objects but also as to their character; for example, she would look at her mother and say to herself that this woman (whom she recognized full well) was not her mother, further, that this woman did not really exist. But it was especially on her own personality that her doubts were most keenly focused or concentrated. "I do not know how I live or whether I live at all; I am a body without a soul or a soul without a body, whichever you like; I cannot be dead, for I feel my heart beating, but is this heart mine and who am I? I must be a mechanical animal, the latest creation of science. But an automaton does not think. How

is it that I can think?" Without suspecting the enormity of the proposition, which would horrify the shades of Descartes, she always concluded: "At any rate, I think—but I do not exist."

It would take a long report, such as I hope to make later, to present all the forms that the feeling of unreality assumed in the case of this patient and all the statements that she made on the subject. But I wish to emphasize one feature that I have rarely observed so fully developed: the feeling of unreality as regards one's recollections. This patient, who related in minute detail numerous events of her past life, constantly interrupted herself to insist that she had no memory at all: "I know full well that I do not actually remember . . . I know it wasn't I who experienced all that. . . . These are not things that really happened to me. . . . I am relating all that, but it seems to me that I might just as well recount something else. Perhaps it is a story that I have read, or a tale that I am inventing. . . . It seems to me that I have had no past existence, and yet I know a lot of things about this individual whom you call by my name—also things that others do not know. It is harder to understand than the mystery of the Holy Trinity."

It was this feeling of the unreality of things, persons and memories which formerly, in the beginning of her trouble, suddenly overwhelmed the patient in the midst of some activity and brought on the disorders that she was unable to explain. At first this feeling quickly disappeared after a little time; then it became more prolonged and finally continuous.

It was this perpetual doubt, this feeling of unreality in regard to all objects, all persons, her own self and the recollection of all past events that constituted the essential mental disorder underlying this peculiar sleep lasting several years.

INTERPRETATION OF SYNDROME OF UNREALITY

While agreeing with the patient that the whole thing is very complex, we know that this syndrome of unreality is rather well known and has been variously interpreted. Although I cannot now enter into a discussion of these theories, I may remark that the present case again confirms the interpretation which I proposed long ago.[2] As I have repeatedly stated, it does not seem possible to ascribe this sense of unreality to modifications of the elementary sensations or perceptions, even though one has in mind more particularly muscular or visceral sensations (myopsychic or somatopsychic). Like all such patients, she presented absolutely no disorder of the elementary sensations. All muscular and visceral sensations were normal. One should not

2. Névroses et idées fixes **1**:40. Footnote 1, pp. 282, 299. La tens in psychologique ses degrés, ses oscillation, Brit. J. Psychol., Medical Section, July, 1821, p. 214.

try to explain an observed symptom by suppositious symptoms assumed for the needs of argument. I am always inclined to believe that there is a close relation between disorders of the sense of reality and disorders of volitional activity, which in this patient were predominant from the start. In her case we find all sorts of abulia, timidity, indecision, doubt, disinterest, impulsiveness, need of being directed by others, etc. . All these disorders followed performance of the slightest task or any prolonged slight effort. Even at the present time, when she appears to be wide awake, I need only direct her attention a little more closely to what I am saying and force her to concentrate an instant, to precipitate a flood of doubt or anxiety or a convulsive attack and to cause her to fall again into deep slumber. While in this brief summary of the case I cannot enter upon a discussion of the point, I am inclined to think that the sense of unreality does not depend on impairment of elementary sensations or elementary psychologic phenomena, for I find no such impairment, but that it depends rather on a reduction of activity, a lack of interest in activity and especially on disturbance of deliberate action, which is, indeed, the very starting point of what we call reality.

Reflection or deliberation is a higher form of integration of behavior; it is an integration of ideas instead of reflexes; it effects a synthesis of the various tendencies awakened, combines them into a definite line of conduct which gives us unity and permanence. For us, individuals are real in proportion to our awareness that we love them or hate them; it is the emotional notion or concept that makes the real. Deliberative action, when it can exist, transforms perceptions. It is not sufficiently recognized that perceptions vary with the different levels of psychologic tension. At the level of reflection the tendencies awakened by a known object are at one and the same time numerous and unified. We are conscious of a stable attitude toward every such object. The same is true of our memories at the level of reflection. We know that we shall always affirm them in the same way. On the other hand, she is never sure of her attitude toward the object that she sees and about which she endeavors to reflect; she doesn't know whether the reminiscence affirmed now will be reaffirmed later. She even indulgently informs one that the very next minute she may assert just the opposite. It is this weakness, this ready exhaustion of her reflective power, that causes her perpetual doubt and her sense of the unreality.

FURTHER CONCLUSIONS

All this may seem far indeed from the slumber of our patient, a sleep which has lasted for five years and which seems to be the essential feature of her trouble. Of course she sleeps, and sometimes

the sleep is more profound and more prolonged than normal. But I do not believe that her state during the last five years has been constantly one of sleep. This young woman appears to sleep much more than she really does. Does she then, pretend to sleep? One may perhaps, call it simulation, if at the same time we concede that the young woman does not in the least prevaricate. She deceives herself and is the first one to believe in her sleep. It is a case of irrational conviction, which is frequent among psychasthenics, in whom the feeling of inadequacy and the obsessions frequently engender delirious states whenever psychic activities are depressed below the level of reflection. Before the Psychopathological Association in Boston, I presented the interesting case of a psychasthenic with obsessions who, from time to time, developed a delirious state characterized by crude or elementary affirmations. I am inclined to believe that these delusions of inactivity, of fatigue, of hallucination, of dementia and of sleep, play a more significant rôle than is generally recognized.

This patient is also a psychasthenic with obsessions, devoid of will power and of the power of reflection. She doubts everything and suffers from a feeling of unreality, at first momentary, later permanent. She gradually became detached from real life and lost interest in it, and that just when she had a fear of the future—fear of life itself. "Life is all a hoax," she said. Little by little she took refuge in delusions and in sleep, which first occurred in transitory fashion during her attacks of depression. When the depression became more profound she began to believe in her continuous sleep. As she doubted everything, she acquired the delusional conviction of her sleep and insanity. This it was that has held her in the pose of continuous sleep for five years.

If these conclusions are correct, this disorder is curable, for states of depression at this level rarely are permanent. By means of the stimuli already noted, I have induced the patient to speak and to do more and more. During the last year, progress has been particularly marked. First, her sleep became less profound, it was easier to awaken her and to keep her clean, to get her to feed herself, to dress herself, and to walk about. For the last six months she has been living with her own family. She is no longer actually somnolent except during the forenoon. During the afternoon she has developed considerable activity, not quite normal of course, but nearly so. The feeling of unreality has remained about the same. This was, I think, the fundamental symptom and is therefore the most persistent.

This curious case of psychasthenic depression with doubt and fear of life, producing a delusion of sleep and the appearance of sleep for five years, shows us, as I have said, how interesting psychologic interpretation may be in nervous disorders.

ABSTRACT OF DISCUSSION

DR. HUGH T. PATRICK of Chicago said that apparently Professor Janet had stopped at a point at which the patient believed that she was asleep and did not exist. Evidently that was his answer to the question why she apparently was asleep and why she acted as if she had lost reality. Dr. Patrick believed there was one additional step to take. Why did she finally believe that she was asleep, and why did she finally believe that she had no existence? It was because she wished to be asleep, and because she wished to have no existence. This obviously bore also on the prognosis, the curability and the therapeutics. In other words, such a patient would promptly and reasonably get well when conditions were such that she ardently wished to be alive, to exist, and wanted to be awake.

The therapeutic measure, then, should be to bring about such conditions that the reaction would not be this defense reaction of creeping into a hole of unconsciousness, but that the patient would wish to come out into the world as it is and partake of the general troubles which we all more or less enjoy.

DR. FOSTER KENNEDY of New York asked Dr. Janet whether he did not consider hysteria invariably as a refuge, as a temporary and inadequate solution of a problem to which the organism could not properly react or solve.

In olden times our forefathers would retreat to a marsh or desert, there to escape the woes and troubles of a tempestuous world; later they saved themselves in monasteries from having to adapt themselves to a difficult environment, and he thought perhaps Dr. Janet's patient had effected a modern solution of an ancient problem.

PROFESSOR JANET of Paris stated, in answer to the question as to whether there were during the years of loss of sense of reality any periods of what might be called real sleep, that on several occasions the patient exhibited periods of profound sleep, which was deeper than normal sleep.

In regard to Dr. Patrick's question, Dr. Janet felt that such patients were really incapable of normally desiring anything—either the desire of getting better or the desire of taking refuge into a state of sleep or nonexistence. He felt this was a sort of automatic refuge rather than one that was desired.

THE SUCCESSFUL REMOVAL OF BRAIN TUMORS

REPORT OF A SERIES OF EIGHT CASES *

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In presenting for review this series of tumors of the brain, successfully removed, we have selected tumors of various types and various localities as representing different problems in localization and in surgical management. In consideration of the operability of brain tumors, the reports in current literature are frequently misleading. For example, many cases are recorded as brain tumors in which the brain tumor was not seen at operation or verified at necropsy. For practical considerations such reports are of no value as neither the nature of the tumor nor its precise location is specified. It cannot be classified on the basis of its pathology nor with relation to accessibility since its precise location remains undetermined. These two phases of brain tumor, character and location, are the only ones of practical moment when considering the question of operability.

Then, too, in estimating the incidence of operable in contrast with inoperable tumors, the time has come when a classification should be made and adopted as a basis of all discussions. It is quite as improper to include all kinds of tumors of the brain in one group when attempting to evaluate operative possibilities or operative risks as it would be to discuss under one heading all surgical lesions of the stomach. There is as much difference between the sharply defined endothelioma and the subcortical infiltrating glioma as there is between sharply defined gastric ulcer and the diffuse carcinoma. In the stomach the latter represent separate and distinct problems; quite as separate and distinct are the problems presented by various tumor types of the brain.

The following classification might be acceptable.

Pretentorial

(a) Benign Tumors

1. Fibroma
2. Osteoma
3. Enchondroma
4. Tuberculoma
5. Endothelioma
6. Miscellaneous

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* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

(b) Malignant Tumors
    1. Glioma
        (a) Cortical, subcortical
        (b) Infiltrating, circumscribed.
    2. Endothelioma
    3. Tuberculoma
    4. Miscellaneous
(c) Pituitary Lesions
(d) Miscellaneous

Subtentorial
    1. Acoustic Tumors
    2. Angle Tumors (Not Acoustic)
    3. Hemispheric Growths
        (a) Endothelioma
        (b) Fibroma
        (c) Glioma
        (d) Tuberculoma
        (d) Miscellaneous

This classification would afford a basis for intelligent discussion and for the comparative studies of reports from various sources. It is manifestly unfair and misleading in considering the question of operative risk to consider tumors of all types together. For example, operations even though only exploratory, in cases of subcortical glioma are always hazardous; and the general mortality of surgical efforts in brain tumor should be reckoned for each of the several groups as proposed in our classification.

Not until, as a class, operations are undertaken at an earlier period will there be any striking change in the incidence of successfully extirpated growths. In a certain number there is an extraordinarily long latent period even though the tumors are not situated in the silent areas of the brain, but in too many instances has there been an unconscionable delay in recognizing tumor probabilities and recommending surgical intervention.

The following cases have been selected as representing tumor types and various locations:

1. An endothelioma of the left occipital lobe
2. A tuberculoma of the parietal lobe near the motor cortex
3. An encapsulated glioma of the motor area
4. A calcified endothelioma of the falx
5. An unusually small endothelioma near the motor cortex
6. A fibroma of the posterior fossa origin undetermined
7. A glioma of the cerebellar hemisphere
8. An acoustic tumor (fibroma).

Two of these cases have clinical features of unusual interest. Case 2 has been employed for a discussion of epilepsia partialis continua affecting the face, and its relation to convulsive movements of the face from irritation of the facial nerve. Case 5 is of special interest because of the small size of the tumor and the importance of aphasia as a localizing symptom. This was a case in which removal of the tumor effected a complete disappearance of all symptoms, a cure in every sense of the word. There has been so much dispute regarding the importance of Broca's area in motor aphasia that Case 5 with sharply defined attacks of motor aphasia and the discovery and removal of a small endothelioma from or very near Broca's area with complete disappearance of these attacks of aphasia is of importance in the controversy regarding aphasia.

REPORT OF CASES

CASE 1.—*Summary: An endothelioma of the occipital lobe removed from a patient, who continued free from recurrence three and a half years after operation.*

History.—M. P., aged 41, was sent to the neurosurgical service of the University Hospital by Dr. R. S. Hooker, as a case of brain tumor. Her previous medical, family and social history contains nothing that bears directly or indirectly on the lesion under consideration.

Four months ago the patient began to complain of frontal headaches, at first observed in the morning, of a few hours' duration. Gradually they increased in severity and continued longer until at the present time they are practically constant. Almost contemporaneous with the headaches dizzy spells developed. These came and went with the headaches. On only one occasion was there complete loss of consciousness and then only for a few moments. With the headaches and dizziness the patient noticed that she was having difficulty in "walking straight" in the dark and more particularly in keeping her equilibrium when going down stairs. Later even in daylight hours she could not walk without staggering. There had never been, so far as she knew, any weakness of either the upper or lower extremities; there had been for several months an aching pain in the legs, especially at night, and edema of the legs coincident with dyspnea and palpitation on exertion. For three months she had been annoyed by vomiting, sometimes once, sometimes three or four times a day, without relation to meals. She thought there had been some diminution in the acuity of her sense of taste, of smell and hearing and some impairment in memory. During the period of her illness she had lost twenty pounds.

Physical Examination.—Cranial Nerves: None of the cranial nerves were involved with the exception of the optic nerve. (See ophthalmic report.)

Motor Function: There was no impairment of movement or strength in either of the upper or lower extremities.

Sensory Function: There was no impairment of any form of sensation, no paresthesia, no loss of sense of position or stereognostic perception.

Reflexes: Biceps and triceps reflexes were equal and normal. The right patellar tendon reflex was more prompt and of slightly greater intensity. There was no ankle clonus and no Babinski sign.

Coordination and Equilibrium: There was no ataxia in the upper extremities. Diadokokinesis was good in both hands. A suspicion of asynergia was aroused by the right finger to nose test. Standing with eyes closed the patient swayed slightly, chiefly to the right.

Ophthalmic Report: The pupils reacted sluggishly to light and accommodation. Vision in the right eye was 6/60; papilledema, plus 5 diopters. Vision in the left eye was 6/30; papilledema, plus 4 diopters. There was right lateral homonymous hemianopsia.

Ear Report: Labyrinths, auditory nerves and brain-stem appeared normal. Bárány tests suggested pressure over the cerebellum of supratentorial growth.

Roentgen-Ray Report: The posterior clinoid processes were somewhat attenuated. The sella turcica was slightly enlarged. There was a possible suprasellar growth. The right internal auditory meatus was larger than the left.

Miscellaneous: The Wassermann reaction of the blood and spinal fluid was negative. The spinal pressure was 32 mm. of mercury, and the spinal fluid contained 10 cells. The urinalysis was negative. Hematolysis: hemoglobin, 60 per cent.; white blood cells 9,900; red blood cells 3,420,000.

Summary of Findings: Headache, nausea, vomiting; right lateral homonymous hemianopsia; papilledema; disturbed equilibrium and doubtful asynergia (right).

Operation.—A craniotomy was performed with total extirpation of the tumor. Ether anesthesia was given, endopharyngeal method, in the face down position. A large cranial flap was reflected to uncover the entire left occipital and in part the left parietal lobe. The dura was found to be under tremendous tension. An attempt at puncture of the posterior horn of the ventricle failed. An attempt to evacuate the ventricle by callosal puncture also failed. Simultaneously an incision was made in the dura and a lumbar puncture performed. On withdrawal of a small amount of fluid excessive pressure was controlled. The tumor presented on the mesial surface of the occipital lobe and extended to the base. Differentiation between the brain tissue and tumor was not difficult, and the tumor was enucleated without difficulty. Encapsulation was complete, and there were no attachments except along the surface of the falx. There was moderate bleeding from the falx; this was readily controlled by hot tampons and muscle grafts. The removal of the tumor left a large cavity bounded by the falx, tentorium cerebelli and occipital lobe. The wound was closed (Fig. 1).

Blood Pressure Observations: During the preliminary steps the systolic pressure was 90; on exposure of the tumor the systolic pressure was 80; after removal of the tumor the systolic pressure was 70.

Pathologic Report.—The tumor was well encapsulated and the capsule was intact throughout except at the point of attachment to the falx. The tumor measured 10 cm. in the largest diameter. The histologic diagnosis was endothelioma.

Subsequent History.—The postoperative convalescence was uneventful. The patient was relieved of all her subjective symptoms, her fields now are partially restored, and she is now well and free from recurrence three and a half years after the operation (Figure 1).

Comment.—This case is cited as illustrative of the type of endothelioma growing pretentorial. The rate of growth is invariably slow,

and there are well authenticated cases in which the history would imply a period of fourteen years' growth. In this series is included a case in which the symptoms were of ten years' duration, and the tumor must have had its origin a number of years before its existence was suspected. It is interesting in the case we are discussing to note that, in a tumor of that size (diameter, 10 cm.) there had been no subjective or objective evidence of its existence more than four months before its removal. The most plausible explanation for this really sudden onset of symptoms is ventricular distention. There is no doubt that at this time the outlet to the ventricles became obstructed, and for the first time signs of excessive intracranial pressure manifested themselves.

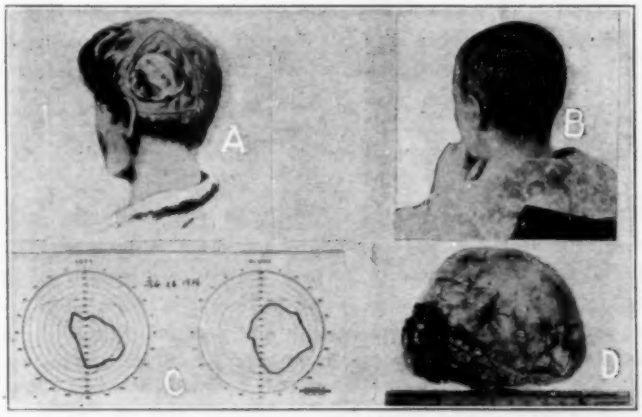


Fig. 1.—*A*, schematic drawing of tumor in situ; *B*, patient two weeks after operation; *C*, right homonymous hemianopsia; *D*, tumor, showing complete encapsulation except at point of origin from falx.

Failure to reach the ventricle on the side of the tumor, either directly or by callosal puncture, would lead one to the conclusion that the ventricle had been compressed and the space practically obliterated by the tumor.

The reason d'etre for some of the symptoms recorded in the history is not clear. Subjectively the patient reported diminution in acuity of sense of taste, of smell and of hearing, but on examination these apparent disturbances were not verified. Taste, smell and hearing were normal.

Had it not been for the homonymous hemianopsia, obviously the characteristic focal symptom, and that at a slightly earlier period the fields were normal, there might have been an error in localization; there was unquestioned evidence of disorder of the right cerebellar hemisphere, the result of the transtentorial pressure of the tumor. It is worthy of note in passing that from the ear clinic the report was

returned without any explanatory note that the examination suggested pressure on the cerebellum by a supratentorial lesion. In the words of the ophthalmic report, there was a right homonymous hemianopsia. This is not strictly accurate; the charts portrayed what was really a quadrinopsia. Not much more than a quarter of the fields was involved. It has been said that a quadrinopsia is peculiar to lesions of the temporal lobe. Perhaps this may be an exception to the rule.

There is little to be said by way of comment on the purely surgical aspects of this case. With the localization clearly defined to the occipital lobe the tumor was readily uncovered by the reflection of a flap large enough to include all the occipital and a part of the parietal lobe. In all exposures for lesions posterior to the Rolandic fissure, the patient should be in the prone position on the operating table, that is, with face down. Our extension head-rest designed originally for suboccipital operations, meets all the indications for supra-occipital cases. The encapsulation of the endothelioma facilitates the operator in finding the line of cleavage between the tumor and brain tissue. The problem of hemostasis arises only when separation of the tumor from its area of attachment or point of origin on the falx begins. Sharp hemorrhage is then encountered and may be temporarily controlled by tampons of cotton or, if the area be not too large, by direct pressure with the finger. The latter is preferable as it lends itself to the "postage-stamp" method of control, which originated during the war, for gunshot wounds of the longitudinal sinus. A piece of muscle or aponeurosis, conveniently taken from the temporal muscle, of proper dimensions, is placed on a finger tip of the free hand and, as the finger of the hand controlling the hemorrhage is withdrawn, the finger with the muscle graft takes its place. When hemorrhage was uncontrolled except with the pressure of a large cotton tampon, the tampon was left in place, the wound closed completely and in the course of three or four days reopened and the tampon removed without recurrent bleeding.

The subsequent course of events is as one would anticipate after a radical removal of a tumor of the endothelioma type. There has been no recurrence or signs of recurrence after a period of over three years. Though of larger dimensions than corresponding tumors of the spinal cord, the surgical possibilities closely parallel one another. The endothelioma of the spinal cord and of the brain when pretentorial is distinctly an operable growth in the technical sense of the word and is one of the most favorable tumors for operation. If not removed entirely, however, it may recur and in the recurrence the tumor is not likely to be well defined. An infiltrating endothelioma is distinctly a malignant growth, it is inoperable and of rapid development.

The absence of word deafness and word blindness in a tumor of the left occipital lobe is to be explained by the fact that the tumor was on the mesial side of the cerebral hemisphere, and evidently originated from the falx cerebri to which it was attached. It had its origin from a region of the brain in which the only localizing sign to be expected was hemianopsia. An endothelioma grows very slowly and causes atrophy of the brain proportional to the size of the tumor and therefore may not cause symptoms until it has reached a considerable size unless it is situated in a part of the brain functionally active.

CASE 2.—*Summary: The removal of an unusually large tuberculoma from the left hemisphere of an adult, male. The operation performed in two sittings and a ventriculogram made in the interval; recovery.*

History.—U. F., aged 26 years, was referred to the neurosurgical service for operation by Dr. William G. Spiller. There was no history of tuberculosis in the family. The patient had been a noncommissioned officer in the United States army, and had served in France. Before his present illness he was engaged as a hotel porter.

He was perfectly well until nine months before coming under observation. While sitting and talking he said he felt sick and suddenly began to make a grunting noise. His head jerked rapidly back and forth. His eyes were starey, he frothed at the mouth, bit his tongue and voided urine. This attack, in which his legs and arms did not move and he did not fall from the chair, lasted several minutes. He was put on the couch and slept for an hour. For seven months he had similar attacks as often as once a week; he knew when they were coming on and carried a spoon which he put in his mouth so that he would not bite his tongue. Once an attack came on while he was walking to a chair; his head began jerking and, though someone was holding him, there was no tendency to fall. For the past two months the attacks have been mild, and he has not frothed at the mouth or bitten his tongue.

Three months ago he began to have trouble in speaking. He knew what he wanted to say but could not find the word. This irritated him so that he often gave up the attempt to talk. He also stuttered. For the past two months he has been drowsy and continually yawning; he complained of headache which he could not localize; occasionally he has been incontinent both as to bladder and bowel. He has dragged his right foot for some time but not markedly so; he was too weak to lift a chair but could drag it where he wished.

Before his illness he was alert and intelligent and had always done well at work. During the past two months particularly there has been a notable change in his condition.

Physical Examination.—The patient was a well nourished and well developed adult negro. He was stuporous and fell asleep two or three times during the examination. If a question was asked abruptly he answered it, but if followed immediately by a second question there was no response.

Thorax: There was no cardiac enlargement but there was a loud systolic murmur at the apex not transmitted to the axilla. The right side of the chest was slightly more prominent than the left, the sternum inclining to the left. The percussion note was resonant throughout; at the left apex there was prolongation of expiration. There were no râles.

Cranial Nerves: There was bilateral papilledema plus 4 diopters, of the second cranial nerve. There were many retinal hemorrhages. Third, Fourth and Sixth Nerves: The pupils were equal and reacted sluggishly to light. The ocular movements were full in all directions. There was no nystagmus. Seventh Nerve: He did not draw up the right corner of the mouth as well as the left. Frequent rhythmical twitching was observed confined to the right side of the mouth. Twelfth Nerve: Slight deviation of the tongue to the right when protruded was noted. Extremities: The grasp of the right hand was weaker than that of the left. In walking he was uncertain and dragged the right foot.

Reflexes: Biceps and triceps tendon reflexes also patellar tendon reflexes, right and left, were not obtained. The Achilles tendon reflex on the left was absent. The Achilles tendon reflex on the right was very weak.

Dr. Spiller reported: The patient is markedly stuporous but can be aroused and can answer questions correctly. During the examination, he has frequent rythmical twitching confined to the right side of the mouth. This is usually more pronounced when he separates his lips in showing his teeth. He does not draw up the right corner of the mouth as well as the left. It is impossible to test him for hemianopsia because of blindness, but he seems to see the hand in the right fields, but it is impossible to determine this accurately. The tongue deviates a little to the right when protruded. The jerking of the right corner of the mouth is like that of epilepsia partialis continua. He responds to all simple commands and answers in short sentences without any definite aphasic symptoms. He understands all that is said to him, so he is not word deaf. The grasp of the right hand is distinctly weaker than that of the left. The finger to nose test is well performed on both sides. It is impossible to test his sense of position because of his mental condition. In walking he is very uncertain and drags the right foot. I cannot obtain either the patellar reflex or the left Achilles reflex; there is a very weak right Achilles reflex.

Diagnosis: Probably brain tumor of left motor region of cerebrum. It is impossible to localize with accuracy because of the advanced condition of the disorder. The continuous jerking of the right side of his face is suggestive of irritation of the face center of the left motor cortex. It is singular that the right side of the face when the patient is quiet may show no tremor but voluntary movement of mouth, as in showing teeth, at once starts it.

Miscellaneous: The blood contained 4,880,000 red blood cells, 11,800 white blood cells and 90 per cent. hemoglobin. The Wassermann test of the blood was negative. Urinalysis: Specific gravity 1.013, alkaline reaction, mucus + +, no red blood cells, from 5 to 10 white blood cells to the field.

Summary of History and Symptoms: History: (1) convulsive seizures for nine months, confined to the head, involving especially the face; (2) aphasia, three months' duration; (3) headache, drowsiness and incontinence for two months, and (4) weakness of the right arm and leg. Symptoms: (1) frequent rhythmical twitching of the right side of the face; (2) weakness of the right side of the face, upper and lower limbs; (3) both patellar and left Achilles reflex absent, right very weak; (4) headache and drowsiness; (5) papilledema, plus 4 diopters bilateral, and (6) pyuria.

Preoperative Diagnosis: Tumor left motor cortex, near face center.

Operation.—A craniotomy was performed by Dr. Frazier and his staff. At the first sitting ether anesthesia was used, endopharyngeal method. A cranial flap was reflected for exposure of the left motor cortex. When the dura was exposed it was found to be under tremendous pressure. Attempts made to

reduce pressure by ventricular puncture on the left side, both direct and by callosal puncture, failed. As it did not seem wise to open the dura until the pressure could be reduced, an attempt was made through separate opening to evacuate the right ventricle. A few cubic centimeters of blood-stained fluid escaped, but the pressure was not affected. At this point the exploration was discontinued and the wound closed.

Ventriculogram.—One week after the first sitting 100 c. c. of fluid were withdrawn intermittently and 100 c.c. of air injected (10 c.c. of fluid withdrawn and 10 c.c. of air injected). The roentgenogram of the skull did not reveal any air shadow in the ventricle. The latter was obliterated evidently by the tumor.

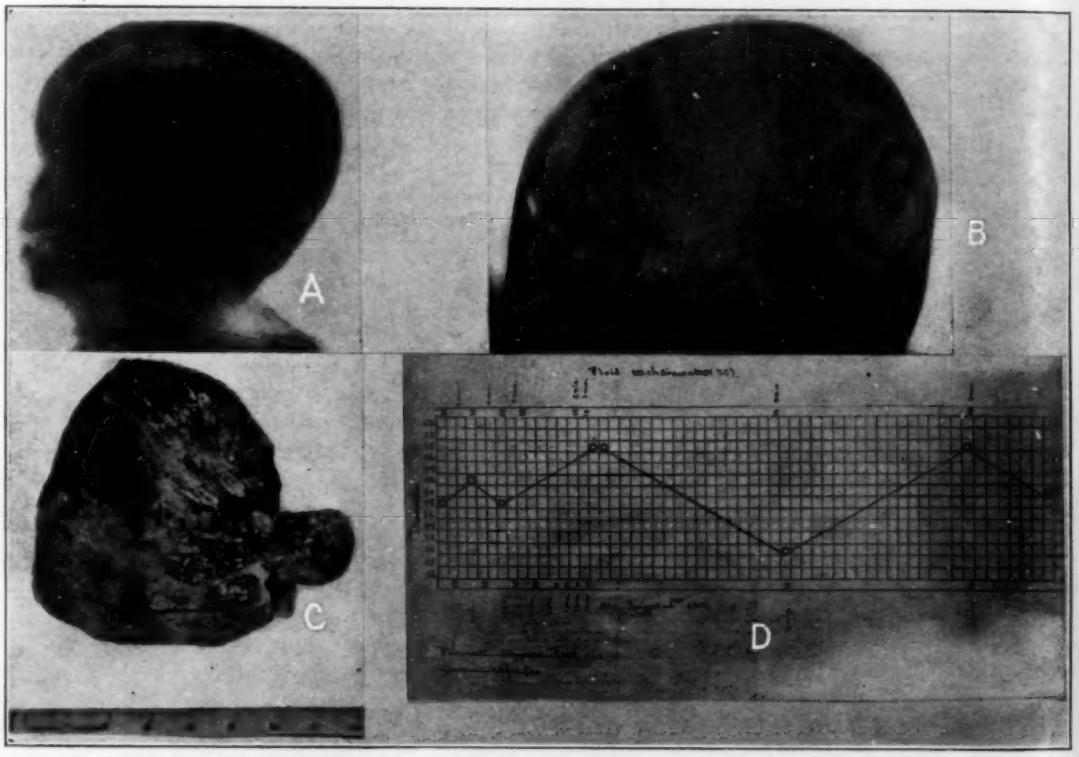


Fig. 2.—*A*, air shadows symmetrical in dilated ventricles of a case of hydrocephalus; *B*, absence of air shadow, ventricles compressed by tumor; *C*, encapsulated tuberculoma which caused collapse of ventricles—photographed immediately after removal; *D*, pressure observations as fluid was withdrawn and air injected.

Exploratory Craniotomy; Second Sitting.—Two weeks after the first sitting and one week after ventriculography exploration was continued. The wound was reopened. The subdural tension was as high as at the first sitting. Lumbar puncture was done with withdrawal of 45 c. c. of cerebrospinal fluid. Immediately tension was restored to normal limits. A dural flap was reflected and the tumor presented at the anterior, inferior quadrant of the opening (near the

junction of the Rolandic and Sylvian fissures). The tumor was sharply defined from the surrounding tissue, and its isolation and extirpation were readily accomplished. The tumor had no relation with the falx and was moderately adherent to the dura, perhaps had already infiltrated it. Oozing from the tumor cavity was trivial and was readily controlled with hot saline irrigations. The wound was closed. As a precautionary measure, 250 c. c. of citrated blood with 250 c.c. of saline solution were transfused at the conclusion of the operation.

Pathologic Report.—On removal the tumor measured 6 by 8 cm. It was irregular in shape, nodular on the surface and definitely encapsulated, except on a portion of the anterior surface. Pathologic diagnosis: tuberculoma (Spiller).

Postoperative Record.—Immediately after the operation there was a transitory hemiplegia and aphasia. Within a comparatively short time the man could move the arm and leg and could talk. Five days after removal of the tumor an examination of the eyegrounds showed a subsidience of the papilledema from plus 4 diopters to plus 2 to 3 diopters, and the signs of hemorrhages seen before the first operation had entirely disappeared. There was for several weeks an irregular febrile course that could not be accounted for and something seemed to retard the patient's convalescence.

A complete search was made for the primary tuberculous lesion. Dr. Pepper reported that there was no evidence of pulmonary tuberculosis. The urine, however, showed an increased quantity of pus, there was tenderness over the region of the right kidney and muscle rigidity in the upper right quadrant of the abdomen. A provisional diagnosis of pyelitis of the right kidney was made and the patient was transferred to the urological service. The patient died while in this service twenty-two months later.

Comment.—A word might be said about the specimen. It was interesting and unusual from several aspects. Tuberculoma is more common in children, is more frequently found in the cerebellar hemispheres, and is not of large dimensions. In all these particulars the tumor did not conform to type. It developed in an adult, in the left cerebral hemisphere and measured over 8 cm. in one of its diameters—a large tumor. Tuberculoma of the brain is secondary to tuberculous lesions elsewhere. The original lesion may be latent or, as in this case, unrecognized before the cranial exploration. If the patient had tuberculosis of the kidney it could not have been very active for a long period, since the patient was reported in excellent health up to within nine months of his operation.

The method of exposure and of extirpation departed from the routine in two particulars. The operation was divided into two sittings and in the interim a ventriculogram was made. When uncertain as to the finding of a tumor or of one's ability to remove it if found, and when the intracranial pressure is extreme, we dislike to reflect the dural flap until at least an attempt is made to relieve the pressure by withdrawal of cerebrospinal fluid. Puncture of the anterior or posterior horn of the ventricle, according to the location of the opening, should be tried. We failed to reach the posterior horn of the right or left ven-

tricle, either directly or by callosal puncture. Either the approach was misdirected or the ventricles were collapsed. We could have proceeded at once to a lumbar puncture but recognized the incidental risks and decided to determine by ventriculography whether one or both ventricles were distended or collapsed and if distended what their geography was.

After one week's interval the ventriculogram was made and the roentgenogram explained our failure to find the ventricles. The ventricles were collapsed, at least there was no air shadow after 100 c.c. of air had been injected. The preparation for a ventriculogram is not unattended with risk under any conditions and particularly when the intracranial pressure is at a high level. This was realized, but one has to measure and weigh relative values, and a complete survey of the cerebrospinal space seemed desirable before the dura was finally opened. The risks were minimized in preparing for the ventriculogram by withdrawing the fluid intermittently. As from 5 to 10 c.c. of fluid were withdrawn, an equal amount of air was injected. Pressure observations were made frequently with a mercury manometer. Using, as we do, a three way cock, merely by the turning of the handle one may (1) take pressure, (2) withdraw fluid or (3) inject air. By repeated observations on the pressure and fractional withdrawal of fluid we reduced the risks to a minimum. There is an appreciable difference in the risk of fluid withdrawn by lumbar puncture in tumors above and below the tentorium. Below the tentorium we regard it as prohibitive.

Ventriculography is in its infancy; as time goes on we will be able to define with greater accuracy the margin of safety and with the confidence of experience will be able to determine its sphere of limitation. In the case under consideration it served a useful purpose. It was of no assistance in the localization of the growth, but it visualized the condition of the ventricles. Had the ventriculogram been made before the operation, we would have been able to finish at one sitting.

Some might question the propriety of a lumbar puncture at the time of the operation in pretentorial growths, but when simultaneously the pressure is relieved above by the reflection of the dural flap, the conditions responsible for foraminal hernia following lumbar puncture do not prevail.

In intracranial procedures mastery of the problems of pressure is essential to the safe conduct of the case. It is in this aspect of the day's work that judgment and experience count.

The condition was far advanced in this case when the man came under the observation of one of us (Spiller). In addition to the general and focal symptoms of tumor, he had a peculiar constant rhythmical jerking confined to the right side of the mouth, and voluntary innervation increased the jerking, as when he separated his lips in

showing his teeth; he could not draw up the right corner of the mouth as well as the left. Occasionally, as in the morning after the night's rest, this rhythmical contraction of the muscles of the right side of the mouth was not so conspicuous but became marked immediately when he answered a question. It seemed as though the left facial center were under continual irritation, and voluntary innervation of this center increased its irritability. The condition was that of epilepsia partialis continua, like that described by one of us (Spiller) in association with Dr. Edward Martin.[1] This case has been referred to by Dr. George Wilson.[2]

In this paper with Dr. Martin one of us (Spiller) was able to give very few references to the literature on epilepsia partialis continua and none to any observations published in English or French. Oppenheim had made a brief mention of it in saying that cases occur in which during the interparoxysmal period muscular twitchings of short duration and without loss of consciousness are observed in one or another part of the body. It had been described by Russian writers—Koshewnikow, Muratoff, and Bechterew, and it had been mentioned by Bruns. In the paper by Spiller and Martin emphasis was laid on the fact that the movements differ from those of myoclonic epilepsy in that the twitchings are limited to a small portion of the body and are usually more intense than in myoclonia. Orlowski had reported a case of epilepsia partialis continua and Choroschko had written a monograph on the disorder, but Bruns was the only one not Russian who had observed this rare type. There seemed to be no lesion discovered in any case, so that the case of Spiller and Martin was the first in which a lesion was actually found and the first therefore to establish clearly a cortical origin for the spasmodic movements. In this case jacksonian convulsions occurred, but independently of these typical attacks the man had twitching confined to the right side of the face lasting an indefinite period, an hour or more at a time and occurring very frequently. There were periods in which he had none of these spasmodic movements, but the movements were almost constantly present and resembled a tic movement or fibrillary twitching. They were not excessive in their intensity and implicated the muscles about the right side of the mouth, about the right eye, the right cheek, the right ear and the right side of the neck. After persisting for some weeks in the face they ceased in this part and were observed in the right upper limb. The whole right upper limb was displaced in these clonic movements,

1. Spiller, William G., and Martin, Edward: Epilepsia Partialis Continua Occurring in Cerebral Syphilis, J. A. M. A. **52**:1921 (June 12) 1909.

2. Wilson, George: The Diagnostic Significance of Jacksonian Epilepsy, J. A. M. A. **76**:842 (March 26) 1921. (Case 1.)

but the displacement was not excessive, and the spasm consisted of slight twitchings lasting indefinitely as did the previous twitchings of the right side of the face. Dr. Martin operated and small gummas were found in the motor cortex.

A case of epilepsia partialis continua was reported by Burr [3] in 1915.

The rhythmical tremor occurring in the present case of tubercle from irritation of the facial center might be confused with the spasmodic jerking resulting from irritation of the facial nerve, as from a tumor of the cerebellopontile angle. In the former condition the contractions are continuous, there is nothing that can be spoken of as an attack; in the latter condition the contractions occur in attacks and simulate the usual epileptiform facial convulsion from irritation of the facial center; indeed convulsive movements have been attributed to irritation in this part, which in reality were caused by irritation of the facial nerve from a tumor. This occurred in a case studied by Dr. Mills with one of us (Spiller) in 1901. Facial spasms were observed in this case under the care of Dr. Mills, and the notes of this case were employed by Weisenburg [4] in a paper on cerebellar tumors published in 1905. The facial spasm was unilateral and caused the mouth to be drawn forcibly to the left and the left eyelids to be drawn together. Both observers thought a tumor was probably located in the facial center of the cerebral cortex of the opposite side and operation was performed; later a cerebellopontile tumor was found. At that time the diagnosis of cerebellopontile tumors was not so well understood as it is now, and facial spasms occurring in attacks had never been reported by any one as a result of irritation of the facial nerve from tumor of the cerebellopontile angle. This patient was completely deaf in the ear on the side in which the facial spasms occurred, but four years before coming under observation the ossicles of the ear on this side had been removed on account of annoying tinnitus, but without the desired result.

In Weisenburg's Case 2 the spasms were limited to the entire distribution of the facial nerve, these coming on without any warning, being first tonic in character and lasting from half a minute to a minute. The lesion was not found. In another case reported by him in which a tumor was found in the right cerebellopontile angle the upper branch of the seventh nerve had been cut, and there had been some

3. Burr: Am. J. M. Sc. **149**: No. 2, 1915.

4. Weisenburg, T. H.: Diagnosis of Tumors and Other Lesions in the Cerebellopontile Angle, J. A. M. A. **50**:1251 (April 18) 1908; Cerebellopontile Tumor Diagnosed for Six Years as Tic Douloureux, ibid. **54**:1600 (May 14) 1910. New York M. J. Feb. 11 and 18, 1905.

return of power in the distribution of this branch. The man had involuntary twitchings in the middle and lower facial distribution; these were sometimes fibrillary; sometimes coarse and spasmodic and were caused by pressure on the seventh nerve. Spasms of the tongue, apparently chiefly right-sided, were observed in this case.

In a patient studied by one of us (Spiller) in 1915 convulsive movements occurred in the left side of the face lasting only a few minutes. A tumor was found in the cerebellopontile angle, and a similar case has been observed recently.

Cushing [5] reported before this Association in 1916 two cases of facial spasm caused by peripheral irritation of the facial nerve from a tumor of the cerebellopontile angle. One of these cases is described in detail. It had been previously diagnosed as a case of focal epilepsy from a lesion of the contralateral cerebral cortex. He states that in a series of about fifty tumors in this angle this irritation symptom in a corresponding degree had not been observed. He refers to a paper by Stewart and Holmes in which it was said that evidence of direct irritation of the facial nerve rarely occurs though twitching may occasionally be observed. Cushing describes the spasm in one of his cases as a twitching of one side of the face beginning as a blepharospasm of mild degree coming on a few times a day and lasting for a few seconds. The attacks gradually increased in number, intensity and surface extent until the spasmodic movements became very severe, coming on every ten minutes or so and persisting for a full minute or two. They began with a twitching and drawing of the muscles about the eye, and gradually the whole side of the face was pulled over in a grimace and the surface muscles of the neck (platysma) were affected. The patient stated that in a few severe attacks the left arm and leg mildly participated in the attack and that the left arm and hand felt numb on several occasions, but this Cushing thought was an erroneous statement. The facial spasms were precipitated by talking, laughing or excitement. The man had no control over the spasms and no weakness of the facial nerve supply. The spasms had existed for nine or ten years. A cerebellopontile tumor was found at operation. As the man lived, the cerebral cortex was not examined, but the facial spasms gradually subsided after the operation.

There may be difficulty in diagnosing between the convulsive movements produced by irritation of the facial nerve and those from irritation of the cortical center for the face, as in epilepsia partialis continua. The careful study of the case will usually permit the correct diagnosis, especially the association with other symptoms. In the form from corti-

5. Cushing, H.: Convulsive Spasm of Face, J. Nerv. & Ment. Dis. **44**:312 (Oct.) 1916.

cal irritation the twitching is likely to be confined to the lower part of the face, as in Pönitz's case to which reference is made further in this paper, whereas in the form from irritation of the facial nerve the whole distribution of the facial nerve is likely to be affected from the beginning. The differential diagnosis may, however, be complicated.

The facial nerve may be irritated to produce convulsive movements of the face without any paralysis of the facial distribution, as in Cushing's case and one observed by one of us (Spiller), or there may be a statement, possibly erroneous, of convulsive movements in muscles outside of the facial distribution; this leads to confusion. It seems singular that convulsive movements of the face from irritation of the facial nerve occur relatively seldom, and yet the number of cases of tumor of the cerebellopontile angle is considerable.

CASE 3.—*Summary: Accurate localization and successful extirpation of an encapsulated glioma from the left cerebral hemisphere; recovery.*

History.—R. McK., aged 30 years, was referred to the neurosurgical service at the University Hospital by Dr. T. P. Farmer of Syracuse, New York, who realized the possibility of a brain tumor, an opinion subsequently confirmed by operation. The patient had had the usual diseases of childhood, including scarlet fever, and appendicitis and catarrhal jaundice; otherwise his health had been good. He had been married two years; his wife and one child, aged 16 months, were well. He denied venereal disease.

Eighteen months prior to admission the patient had a convulsive attack while asleep. It was noted that during the attack he lay on his face moaning "with the left foot out of bed shaking." In two weeks he had another attack, this one during the day. He lost consciousness and foamed at the mouth. Two weeks later he had another attack, and during the first twelve months he had twelve convulsive seizures. During the past six months the seizures had been more frequent, two or three a day, but of quite a different character. In these he did not lose consciousness; the convulsions always began in the fingers of the right hand; the fist was clenched and the arm abducted to a right angle with the body. The muscles of the right side of the neck were involved in the tonic spasm. He had never bitten his tongue, nor had there been any involuntary evacuation. With the exception of these attacks, he felt perfectly well.

After admission to the hospital, the convulsions continued; there was in rapid succession flexion of the fingers, wrist and elbow. The arm was then raised to a right angle with the body, and the muscles of the neck participated in the clonic spasm. Consciousness was preserved.

Physical Examination.—Thorax: There was slight impairment on percussion at the left apex; inspiration was slightly prolonged and harsh. The area of cardiac dulness was normal; there were no cardiac murmus.

Cranial Nerves: There was no impairment of function.

Motor Function: In all movements the right forearm and right arm were distinctly weaker than the left; there was no difference in strength of movement between the right and left lower extremities.

Sensory Function: This was unimpaired.

Reflexes: The biceps and triceps tendon reflexes were exaggerated on the right side; the patella tendon reflexes were equal and normal. There was no ankle clonus and no Babinski sign.

Coordination and equilibrium were undisturbed.

Ophthalmic Report: The pupils were equal and regular in outline and reacted equally to light and accommodation. There were no ocular palsies and no nystagmus. The fields were not contracted and there was no papilledema.

Miscellaneous: The Wassermann, globulin and colloidal gold tests of the spinal fluid were negative. There were 8 cells per centimeter. The Wassermann reaction of the blood was negative, hemoglobin 81 per cent. and the white blood cells numbered 7,900. The roentgenogram was negative.

Fig. 3.—Encapsulated glioma.

Summary of Physical Findings: The findings were (1) initial convulsive seizures general in character; (2) later substitution of these with typical jacksonian seizures (right); (3) weakness of the right arm, and (4) exaggerated biceps and triceps tendon reflexes (right).

Preoperative Diagnosis: Tumor motor cortex (left).

Operation.—Dr. Frazier and staff performed an exploratory craniotomy. Ether anesthesia was used and a flap was reflected for exposure of the left motor cortex, two-thirds in front and one-third behind the Rolandic fissure. As soon as the cortex was exposed the lesion was seen in the upper half of the field, in front of the Rolandic fissure. Closer examination revealed a sharp line of demarcation between the tumor and the brain tissue. Around the margins of the tumor as exposed the pial vessels were ligated and with gentle dissection the tumor was freed from the surrounding tissue and removed. The tumor extended 5 cm. below the surface and inward as far as the median line. Its point of origin was undetermined, although at the operation we suspected the

falx. Following the removal of the tumor bleeding was trivial; there were but three bleeding points in the tumor bed and these were controlled with muscle grafts. The tumor measured 5 by 6 cm. The wound was closed without drainage.

Pathologic Diagnosis.—Grossly the tumor resembles an endothelioma but histologically it is a glioma, apparently well defined, but not entirely so (Dr. W. G. Spiller).

Postoperative Course.—Immediately after the operation the patient had aphasia and hemiplegia. Within a week, however, he began to recover from both and eventually fully recovered his power of speech and the power of the right leg. The movements of the right arm were still impaired and the limb somewhat spastic. When the patient was examined within a year of the operation, he had had four convulsive seizures; the first two occurred a few months after the operation and were jacksonian in type. There was twitching of the right side of the face and a burning, throbbing sensation in the right arm. In the first convulsion he lost consciousness, in the second he did not.

The second two convulsions occurred nine months after the operation. While talking, speech suddenly failed him, he felt a tense drawing feeling in the right arm and leg, the right arm was abducted and the right leg extended. He remained unconscious for ten minutes. Immediately after he went to bed and within a few moments, a second convulsion, similar to the first, occurred.

Nine months after the operation speech was undisturbed and the power of the leg was restored, but the arm continued weak and spastic. The eyegrounds were normal.

Comment.—The facility with which tumors may be localized when originating in or over the motor cortex is a point in their favor from the standpoint of operability. One might presuppose that in the majority of cases an early diagnosis would be established when the tumor was in its incipiency. Such is not always the case, however. In two of the cases in this series, Case 2 and Case 3, the tumors were of large dimensions and the duration of the symptoms only eighteen months in one and nine months in the other. The origin of these tumors, as to time, is, of course, a matter of conjecture, but as a rule tumors of the brain are slow in growth and one might presuppose with reason a growth 6 cm. or more in diameter might have been several years in process of development.

The history of the convulsions in this case is interesting. For the first twelve of eighteen months prior to the operation they were general in character with loss of consciousness but without focal signs; in the last six months conditions were reversed; consciousness was preserved and the convulsions were typically jacksonian. One can readily account for jacksonian seizures in a lesion pressing on or actually infiltrating the cells of the motor cortex; but in the absence of signs or evidence of increased pressure, without distention of the ventricles, one wonders why the incipient convulsions should have been general and attended with lost consciousness, unless they were caused by general increase of

intracranial pressure before the motor cortex of one side was irritated by the tumor.

As in many pretentorial growths, there were no pressure signs; neither headache nor papilledema. This implies of course that the outlet to the ventricles had not been closed and as yet there was no internal hydrocephalus, the most common cause both of headache and papilledema in tumors of the brain without regard to location.

The pathology of this tumor is of unusual interest. In his report Spiller referred to the growth as having the gross appearances of an endothelioma, and so it appeared to the operator. But the histologic examination revealed a glioma, so that we were dealing with a definitely encapsulated glioma, a lesion quite unique in the experience of our clinic. Not only were we deceived in the naked eye appearance of the growth, but as well in our assumption that it had its origin in the falx. With regard to encapsulation of gliomas, of course one often sees what appears to be a distinct wall surrounding a cyst of gliomatous origin. But in this case there was no cyst, and in all the gliomas hitherto exposed in our clinic there have been ill defined margins as the infiltrating tumor invaded the surrounding tissue. We have never seen a definite encapsulation such as in the specimen under consideration. There was, to be sure, a small area on the surface of the growth without a capsule, but we are disposed to believe that in removal this portion of the capsule had become detached.

The postoperative complications, aphasia and hemiplegia, did not disturb us at first. The trauma and edema incidental to the removal of the brain tumors often cause transitory interruption of function. In so far as the aphasia and leg were concerned, the recovery of function in both was no exception to the general rule. The persistent weakness and spasticity of the arm imply, however, irreparable injury.

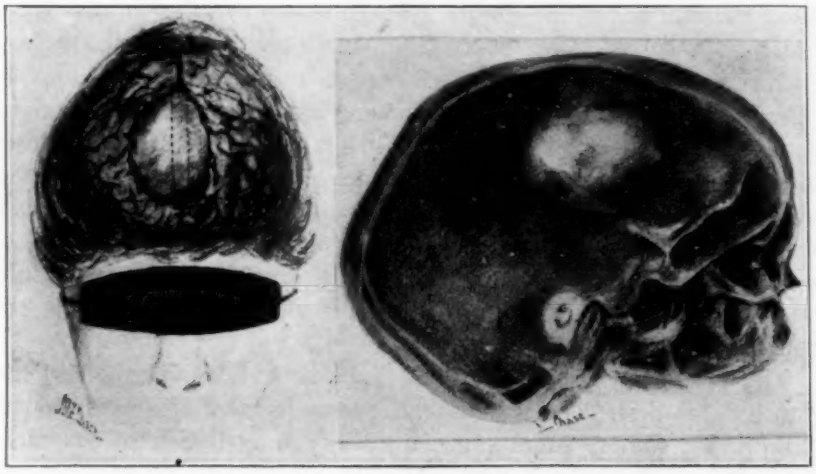
Whether the convulsions subsequent to the operation were caused by a recurrence or by cerebral trauma is still an open question; either of course would explain them; but one would hardly expect a recurrent growth within five months of the operation, the date of the first convulsion, to be of sufficient size to be responsible for them. We can, with just as much reason, attribute the convulsion to the immediate effects of the operation trauma, hemorrhage and cicatrization. In all patients with epileptic seizures we give luminal, 1½ grain, in the evening, from the day of the operation for a period of six months to a year or longer.

CASE 4.—*Summary: A calcified endothelioma intrahemispheric, exposed at operation and treated by radium implantation with relief from convulsions and headaches.*[6]

6. While this tumor was not removed, this case is included in the series because of the relief which followed the exposure of the tumor and direct radium implantation.

History.—O. T., aged 37 years, was admitted to the neurosurgical service of the University Hospital, April 11, 1920, referred by Dr. M. H. Tallman. She had had measles and chickenpox in childhood and diphtheria at the age of 20. She had had two miscarriages, one before and one after the birth of a living child (now 3½ years old). A maternal aunt had epilepsy. Otherwise her family history was negative. She was married five years ago. She was engaged in housework and her living conditions were wholesome.

Ten years before admission the patient first complained of momentary attacks of dizziness, occurring about seven times a day. Three months after the onset of these she noticed that during an attack the left knee became weak and she nearly fell. These attacks continued for four months, at the end of which time she had her first severe convulsion. The convulsions were preceded by a choking



A *B*

Fig. 4.—*A*, schematic drawing showing relation of tumor to longitudinal sinus; *B*, drawing of roentgenogram showing calcification of tumor.

sensation, the left arm was abducted, the lower left limb drawn up, and she lost consciousness for about ten minutes. She came out of the convulsions groaning, but did not fall asleep. Only the left side of the body was involved in the convulsions. At first they occurred about once a month, then gradually they became less frequent and after her first child was born there was an interim of a year and nine months in which she had no attacks at all. About two months before admission the attacks recurred, lasting a few minutes; in these attacks her left upper and lower limb felt numb, and she could not raise her left arm or foot. During this period for the first time she complained of blinding headaches, occurring about once a week, sometimes in the right temporal region and sometimes in both.

Physical Examination.—Examination of the scalp, nares, teeth and tonsils was negative. The post-cervical glands were palpable. The lungs were normal. There was a systolic murmur over the aortic area of the heart. Blood pressure was: systolic 100; diastolic, 70. Examination of the abdomen was negative. Urinalysis: The specific gravity was 1.024. The urine was acid and there was a

trace of albumin. There was no impairment of motion in either arm or leg, and normal sensation was preserved. Both the right and left patellar reflexes were exaggerated.

The Wassermann test of the blood and spinal fluid was negative.

Eye Examination.—Muscular movements were free and equal. The pupils were equal and regular and reacted to light and accommodation. Right eye: The media were clear and the disk margins were veiled with fine striations running out into the retina. The veins were overful and slightly tortuous. There were no discrete lesions of the choroid or retina. Left eye: The media were clear and the disk margins obscured. The disk was elevated from 1 to 2 diopters. The veins were full and tortuous. There were no discrete lesions of the choroid or retina.

Roentgen-Ray Examination.—This showed a calcified tumor, extending from the vertex more than half way to the base of the skull. It was viewed anteroposteriorly, in the midline position, extending more to the right than to the left. There was no evidence of intracranial tension and no sellar deformation.

Preliminary Diagnosis: Calicified endothelioma.

Exploratory Craniotomy.—First Stage: Flaps were reflected on both right and left sides, uncovering the region of the tumor and the longitudinal sinus. The patient's condition was not tolerant. No attempt was made to remove the tumor at this sitting.

Second Stage: Nine days later the flaps were again reflected and the tumor exposed. The situation of the tumor with relation to the longitudinal sinus made it evident that a section of the sinus would have to be removed with the tumor. Furthermore, the tumor had infiltrated the overlying dura so that to extirpate the tumor successfully would require removal en bloc of the tumor, overlying dura and a portion of the longitudinal sinus. A dural incision circumventing the tumor was made. The longitudinal sinus, anterior to the tumor, was divided and closed with silk ligatures. Though there had been little bleeding and a minimum of trauma, the patient's condition gave us concern. The pulse rate was 150 and of poor quality. No further steps at extirpation were taken for the moment. Exploring with the finger the tumor was found to be attached to the falx and to extend almost (if not to) the base of the skull. So firm were its attachments that there was no play either in the lateral or anteroposterior directions. On account of the patient's condition and because the tumor was so firmly attached, further attempts at removal seemed too hazardous. The dural and scalp incisions were closed. Direct transfusion was made of 500 c.c. of citrated blood.

Radium Implantation.—May 14, 1920, eighteen days after operation, under procain anesthesia, a small area of skull overlying the tumor was removed with rongeur forceps. A container with radium, 50 mg., was implanted in the tumor. In attempting to direct the container toward the center of the tumor, it was obstructed by some impermeable substance (calcified areas in the tumor). The container had to be directed forward to imbed it completely in the tumor. The radium was removed in eighteen hours. The dosage was 900 mg. hours.

Postoperative Record.—Immediately after this exploratory craniotomy the patient developed a hemiplegia from which she had largely recovered at the time of her discharge. Thirteen months after the operation, her physician

wrote that the patient was feeling very well, that while she had attacks of dizziness once in a while, she had had none of the convulsive seizures since her discharge. In response to a suggestion that she return for a second radium treatment, the patient stated that in view of her improvement, she did not feel justified in incurring the expense of the journey across the continent from her home in the West to the hospital.

Comment.—There are many points of interest in this case. For almost ten years the history of the growth was characterized solely by unilateral convulsive seizures involving the left arm and the left leg. The fact that after the seizures the patient experienced weakness in the arm and leg had aroused a suspicion of tumor. It was not until well within the tenth year of her illness that other focal signs developed, such as transitory periods of numbness in the left arm and left leg and, for the first time, blinding headaches. In spite of the duration of the lesion and of its huge proportions, in spite of the blinding headaches, there was little evidence of increased intracranial pressure in the condition of the optic disks. It is noted in the eye examination that there was an elevation of only from 1 to 2 diopters in either disk.

Had there been any doubt as to the diagnosis, and there could have been little, the evidence of the roentgenogram was conclusive. From the shadow of the tumor in the roentgenogram one is justified in making a preliminary diagnosis of calcified endothelioma, although in another case, a tumor quite as vividly revealed in the roentgenogram proved to be an enchondroma.

The location of the tumor, practically intrahemispheric and beneath the longitudinal sinus, necessitated a two flap operation, one reflected to the left and one to the right. This two flap operation I found gave a splendid approach and exposure. The immobility of the tumor is difficult to account for, unless it was adherent to the base of the skull, and yet we know that many of these midline endotheliomas take their origin from the falx. Except for this unusual immobility there appeared no obstacles to its removal, and in a more robust patient an attempt would have been made to mobilize it. After the operation it occurred to me that we might have attempted a subcapsular extirpation such as we practice at times in cerebellopontile angle tumors.

In this particular case, however, I doubt whether the patient would have survived any radical undertaking. The intolerance to surgical intervention of patients with tumors of long standing is an interesting speculative study. From the very beginning of an operation, the pulse, as in this case, becomes accelerated; a rate of 140 to 150 is common. The mere reflection of the osteoplastic flaps seemed a sufficient undertaking for one sitting and a two stage operation seemed imperative. At the second sitting, even before any attempt at demobilization of the

tumor had been made, there was reason for anxiety. The disturbance of the cardiac mechanism in brain tumor cases I have attributed, rightly or wrongly, to a delicately balanced blood supply of the medullary centers. How else can one account for the rapidity of pulse rate and the falling of blood pressure? In the absence of serious hemorrhage there apparently is no other way of accounting for them.

CASE 5.—*Summary: Successful extirpation of an endothelioma 3 by 2 cm. from a patient whose illness began with fleeting attacks of aphasia, followed by jacksonian seizures; recovery.*

History.—C. M., aged 17 years, was referred to the neurosurgical service at the University Hospital by Drs. William G. Spiller and H. M. Fussell. When 16 years of age, she first consulted one of us (Spiller) Sept. 21, 1918. About a year previously she suddenly lost the power of speech for about three minutes. while walking along the road. She was frightened. At first she stuttered and then was entirely unable to speak. There seems to have been no other symptom at this time. About a month later she again lost her speech suddenly, for about a minute and a half, without any premonition or stuttering, and again she had no other symptom. She tried to write while she was unable to speak this second time but could not form any letters during this period. She had lost her speech about six times in all for a brief period. About the middle of July, 1918, while in a field, objects suddenly seemed to be going around; she fell to the ground and was unconscious about ten minutes. About a month later, while riding horseback, she suddenly lost her speech; she got off the horse and laid down on the ground and may have lost consciousness. In the later attacks with loss of speech she had a peculiar sensation in her right forearm and hand, and she could not move the right hand at these times, but the hand twitched and the two times she lost consciousness the right side of the mouth twitched.

About three weeks before coming for examination her mother found her making a noise with her mouth, she was somewhat rigid, the right side of the mouth twitched and she was unconscious. In this attack there was a numb feeling in the whole right side, face, limbs and trunk. Dr. Zentmayer in September, 1918, found the veins of the left retina a little fuller than normal.

Attacks occurred which were observed by her parents. They began, the patient said, with a loss of voice, when she would lie down; a twitching of the right arm and the muscles around the mouth was noticed, which were drawn to the right side. In the early attacks she thinks the right leg moved but this is questionable. The attacks occurred about a week apart; the duration was the same, from one to three minutes, and they were characterized by twitching of the right arm and mouth muscles. Each attack began with what she felt was a loss of voice, which forewarned her so that she lay down. In the present month she had had two attacks and the beginning of a third, which apparently was abortive. In these attacks she had held the right hand firmly on her breast and the arm had not appeared to move, but the muscles of the mouth trembled and were drawn to the right. These last two or three attacks had not lasted for more than a minute or two and with less convulsive movements, the arm apparently not taking part.

Repeated examinations of the eyegrounds showed no positive change. Her muscular tone seemed as good as ever, there was not the slightest diminution of power in the right arm, and the grip of the right hand was perhaps unusually

strong. Her general health and spirits, if anything, had been better this month than in the past two. She had as lively an interest in affairs as usual, was planning to return to school, and had been allowed to go to entertainments and take part in occasional dances as usual.

Preoperative diagnosis: Tumor of the motor cortex, left, near or in Broca's area.

Operation.—A craniotomy was performed by Dr. Frazier and his staff. Ether anesthesia was used. An osteoplastic flap was reflected, designed to expose the motor zone and the region of Broca. When the dural flap was reflected there was seen just above Broca's region an area of minute dimensions, less than a centimeter in diameter, that differed in appearance from the normal brain tissue. The nature of it at first was not realized, but on closer examination it proved to be a tumor which without difficulty was separated from its bed and removed. The tumor was quite the smallest of any in the experience of our clinic, measuring in its largest dimension not more than 3 cm. The operation was free from any technical difficulties. The wound was closed without drainage.

Pathologic Diagnosis: Endothelioma.

Comment.—The attacks of loss of speech indicated a lesion of the left cerebral speech area, and they seem to have been purely of the motor type, as no sensory aphasia was noted. At first there was no other disturbance than that of motor speech and writing, and as the tumor was very small and could not have caused much pressure the loss of speech was a sharply defined focal symptom. It would seem to indicate a lesion of Broca's area. The loss of speech was complete temporarily, and this is the form of aphasia to be expected in a lesion of Broca's area. It is very different from that occurring with a small lesion of the sensory speech area. In this improper words are used, the patient cannot think of the words he wants to use and uses others incorrectly. This has been seen several times by one of us (Spiller) in tumor of the left temporal lobe. It is the earliest manifestation of beginning word deafness, and the improper use of words precedes distinct signs of word deafness. Indeed this improper use of words in the early stage of lesions of the left temporal lobe may be so slight that a search must be made for it. A patient now under observation from whom a large tumor of the left parietal lobe has been recently removed by Frazier understood everything said to him and spoke very well, but he at times could not think of the words he wanted to use, especially the names of objects, and showed beginning word blindness in that he confused figures and did not understand everything he read.

The loss of speech in this young girl was not caused by a temporary paralysis of the face center, as it does not appear likely that a temporary paralysis of all the muscles of speech could result from a lesion of the face center of one side. This probably would result only from a bilateral lesion of the center. The agraphia also is evidence of a true motor aphasia.

Transitory motor aphasia with convulsive movements of the right side of the face and right upper limb later constituted the whole clinical picture. The father of the patient was a physician and understood the focal importance of these symptoms. Rather than permit his daughter to continue having these distressing attacks he consented to an operation, with the discovery of a minute well defined endothelioma in or very close to Broca's area, distinctly separated from the underlying brain, and its successful and complete removal. Soon after the operation all convulsions ceased and no more have occurred. The recovery is complete in every detail and there seems to be no probability of recurrence. Not a single symptom persists.

The occurrence of complete transitory motor aphasia in a young person may therefore be a focal symptom of importance. In an elderly person it may result from arteriosclerosis, paresis or some other cause.

A case very similar to this one, except in regard to the aphasia, is reported by Pönitz.[7] Aside from some hysterical manifestation, the important symptom was clonic convulsion of the left side of the face, except the upper part of the face, occurring in attacks, of two years' duration. Later the left upper limb became involved in the convulsions. A solitary tubercle the size of a cherry stone was found in the right facial center.

CASE 6.—*Summary: The successful removal of a tumor, with the characteristics of a fibroma, from the right cerebellar fossa. The patient recovered and continues free from symptoms three years after the operation.*

History.—Mrs. O., aged 45 years, was referred to the neurosurgical service of the University Hospital by Dr. G. A. Young of Omaha, to whom I am indebted for the following history:

Four years ago the patient began to complain of dizziness and unsteadiness of gait; the latter was slight at first but gradually became more pronounced, and it was constant. The dizziness was not associated with rotation of object or self. Eighteen months ago she began to complain of headache and vomiting periodically. Some months later she experienced a sense of numbness in the right side of her face.

At the present time there is a definite hypesthesia with loss of the conjunctival reflex. There is a slight weakness of the right facial distribution but no disturbance in either of the cochlear or vestibular branches of the auditory nerve. The visual fields are slightly contracted and there is an elevation of two diopters in each disk. There is no disturbance of the motion or sensation in any of the four extremities, and the reflexes are normal. There is a suggestion of adiadokokinesis in each upper extremity. She speaks of her left side as being her good side, without being able to say precisely in what way it is the better. At the present time she has comparatively no headache, but nausea and occasional vomiting persist.

7. Pönitz: Deutsch. Ztschr. f. Nervenh. **67:** Nos. 1 and 2, p. 89.

At the present time the Bárány tests show an irregular spontaneous past pointing in both arms which may be either to the right or left; both vertical and horizontal canals react to the caloric test in both right and left ears. In the turning test she shows a normal nystagmus reaction in either direction, the past pointing is somewhat indefinite, but she past points in the right direction when turned to both left and right. The falling reaction is normal also to the right and left. The subjective sense of after-turning vertigo has a duration of about twenty seconds when turned to both right and left.

Preoperative Diagnosis: Tumor of the right cerebellar chamber.

Operation.—A suboccipital craniotomy was performed by Dr. Frazier and his staff. An encapsulated tumor was found and removed from the right cerebellar fossa.

Pathologic Diagnosis: Fibroma.

Unfortunately the record of the physical examination and the operative details have been lost, so that we are able to present only the foregoing facts. The patient recovered and is entirely well, three years after the operation.

CASE 7.—*Summary: Seven years after the onset of symptoms and one year after a suboccipital decompression, extirpation of glioma from left cerebellar fossa; three courses of radium therapy before operation, during which tumor became encapsulated. Three years after extirpation of growth no evidence of recurrence.*

History.—E. E. S., aged 38 years, was referred to me by Dr. Charles F. Neu of Indianapolis. The family, social and previous history were without interest.

Six years ago the patient's wife observed that his gait was shuffling, but no other symptom was noticed until three years later when the hearing of his right ear became impaired. Two and a half years ago he noticed numbness in his lips at the angle of the mouth, and an abrasion on the tongue which he had bitten unknowingly. About the same time the hearing of the left ear became impaired and sensation in the left side of his face was lost. In walking he staggered to left and occasionally lost his balance and fell. His eyesight was not as good as it had been and headaches became more or less constant. During the preceding eighteen months he had attacks of vomiting and, because of his disability, had to give up his position as a school teacher. Within the year and before he came under our observation, a suboccipital decompression was performed. This relieved his headaches in part, but the patient thought his incoordination was more marked. Recently he had had difficulty in talking and swallowing, occasionally choking when swallowing liquids.

Physical Examination.—Head: There was a moderate hernia at the site of the previous decompression.

Cranial Nerves: Second, Third, Fourth and Fifth Nerves: The pupils reacted to light and accommodation. Ocular rotations were full. The vision in the right eye was 6/12; the papilledema plus 5 diopters. Vision in the left eye was 6/15; papilledema plus 6 diopters. The disk was rather spread out and more succulent than one would expect in a choking of long standing. It may imply an acute exacerbation (Holloway). The fields showed some irregular contraction but nothing of localizing significance. There was horizontal nystagmus on lateral rotations; on upward and downward rotation the movements became vertical. Fifth Nerve: There was a sense of numbness in the left trigeminal distribution. Seventh Nerve: The upper branches were slightly involved. Eighth Nerve: There was marked impairment of hearing in the left

ear. There was vertigo on stimulation, with complete absence of past pointing of the left arm to the left. Impaired response from the right or functioning ear was noted. The right labyrinth was not affected. The findings suggested a lesion of the left cerebellopontile angle with extension to the left cerebellar hemisphere and brain stem. Ninth Nerve: There was difficulty in swallowing. Twelfth Nerve: There was no deviation of the tongue when protruded.

Extremities: The grip of both hands was firm and equal. No weakness was present in the lower extremities.

Reflexes: Biceps, triceps and patellar tendon reflexes were more marked on the left than on the right side.

Coordination: Movements of the left arm and leg showed marked incoordination. Adiadokokinesis was marked.

Miscellaneous: The blood pressure was: systolic 135; diastolic 70. The red blood cells numbered 2,510,000; the white blood cells, 8,240; hemoglobin 92 per cent. The urinalysis was negative. Roentgenogram: The posterior clinoid processes were exceedingly thin. The sella turcica was not enlarged. The right and left internal auditory meatus were of equal dimensions.

Preoperative Diagnosis: Tumor of the left cerebellopontile angle.

Operations.—A suboccipital craniotomy was performed by Dr. Frazier and his staff. Ether anesthesia was used, endopharyngeal. On reflection of the flaps a tumor was exposed occupying the entire left fossa. It had the gross appearance of a glioma and contained a large cyst. Extirpation was considered impossible and 50 mg. of radium were inserted into the cavity of a cyst to be removed in eighteen hours. The wound was closed.

Before the patient was discharged, radium was applied by Dr. Pancoast over the surface in the suboccipital region. Three months after the operation the patient returned for observation and a second course of radium therapy. His sight, hearing and articulation were improved. Incoordination was about the same. There was a marked subsidience of the choked disk and the nerve margins were readily discernible. Elevation of the right disk was plus 2 diopters; of the left plus 3 diopters.

Eight months after operation a third course of radium therapy was given. He was totally deaf in the left ear but hearing in the right ear was much improved. There was no particular change in the condition noted except in the movement of the left leg and vision. The heel to knee test (left) revealed no ataxia. Vision in the right eye was 6/9; in the left 6/9 (previous to operation it was 6/20-6/15). The fundi level on either side was plus 1 diopter. The disks were somewhat obscured on the nasal side but were visible in the temporal margins.

Ten months after operation, the patient returned with evident aggravation of symptoms, particularly noticeable in the three previous weeks. Headache had returned, hearing in the right ear again becoming impaired; lately he had been swaying to the right in walking, previously only to the left. His vision was not as good; it was 4/15 in each eye, the disks were plus 4.5 diopters. In other respects there had been no appreciable alteration in the clinical picture. Because of the evident recurrence and at the patient's earnest request a second operation was agreed on.

Operation 2.—A suboccipital exploration was made. When the musculo-aponeurotic flap was reflected, the dura not having been replaced at the previous occipital decompression, the tumor presented itself. But in appearance it differed in that now it was well encapsulated. Complete extracapsular extirpation did

not seem feasible; the capsule was split and the contents removed. Grossly the tumor had the appearance of a cystic glioma. Again 50 mg. of radium were introduced within the capsule, to be removed in eighteen hours. Wound closure was made.

Pathologic Diagnosis: Glioma.

Final Note.—Three years after tumor extirpation and nine years after the appearance of the first symptom, the patient returned for examination. He reported that he had been active in business, and had less difficulty in walking; the hearing in the right ear was much more acute, and he had had no nausea,

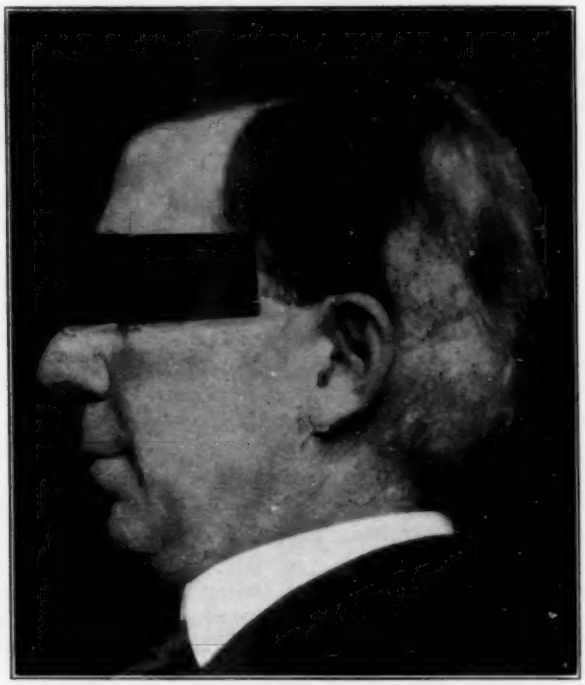


Fig. 5.—Photograph of patient taken nine years after operation. In the area to which radium was applied the hair is white.

vomiting or headache. On examination there was ataxia and dysmetria in the left upper and lower extremity; the nystagmus, particularly in extreme positions, persisted. Vision in the right eye was 6/9 plus 2, in the left 6/12 plus 2. The margins of the disks were clear; no papilledema.

Résumé of Case.—Nine years ago, the first symptom, disturbance of the gait, appeared. Six years ago there was disturbance of hearing followed by numbness of the face, failure of vision, vomiting, headache, difficulty in swallowing and speech, dysmetria, etc. Four years ago at a suboccipital decompression the tumor was exposed but not removed; radium therapy. Three and one half years ago radium therapy (course 2) was utilized. Three and one quarter years ago a third course of radium therapy was given. Three years ago extirpation of the growth was performed and a fourth course of radium therapy was given. At present there is no evidence of recurrence.

Comment.—The larger one's experience with tumors of the brain the more one is struck with their rate of growth. How slow it is compared to malignant tumors elsewhere. Even is this so, as in the case under discussion, with gliomas which in their cellular characteristics resemble the sarcoma group. In this case there were signs of a tumor developing six years before the operation, and though radical extirpation was not possible in the surgical sense, there has been no evidence of recurrence three years after removal.

The localization of the growth was not difficult. In the evolution of the clinical picture the first symptom was disturbance of gait and some time elapsed before there was any other evidence of tumor. The three year interval between the initial symptom and impairment of hearing was more than suggestive of a lesion originating in the cerebellar hemisphere rather than the cerebellopontile angle. At least this interval excluded the likelihood of an acoustic tumor. As a matter of fact, there was never complete nerve deafness. One of the earlier symptoms was a sense of numbness in the face, on the affected side, and yet at no examination could one demonstrate any loss of appreciation of tactile or pain sense.

What may have been the effect of radium therapy on the tumor growth is a matter of speculation. At the second operation there was a definite encapsulation of the growth, not observed when the tumor was originally exposed at the decompressive operation a year before. That radium incites hyperplasia of connective tissue elements is generally acknowledged, but this would hardly explain the formation of a capsule about the tumor, and connective tissue elements within a glioma are negligible. From what we know of the effects of radium on tumors, we have come to the conclusion that it is more effective in the endotheliomas than in the gliomas.

When an entire cerebellar hemisphere has been destroyed by the tumor one cannot of course expect recovery of function after the tumor is removed. There must persist, as in this case, disturbance of the function, attributable to the hemisphere destroyed. And so while the headache, vomiting, papilledema and to some extent the deafness disappeared (all pressure phenomena), the ataxia, dysmetria, disturbance of equilibrium and gait persisted.

As to the operability of gliomas, in many instances they have been regarded by us as inoperable, particularly diffuse subcortical infiltrating growths of the cerebral hemisphere. But in this case the tumor occupied the entire left cerebellar fossa; one could not see a vestige of the cerebellar hemisphere, and as it appeared well encapsulated the contents of the capsule at least could be extirpated. Even should there be a recurrence at some later date, a second operation should be advised with a reasonable hope of removing the recurrent growth.

Prolongation of life, even after partial extirpation, should be given a prominent place when discussing the pros and cons of surgical interference in brain tumors. To be able to prolong the patient's life, in a reasonable state of health and activity and at the same time to conserve vision, is in itself a sufficient endorsement of surgical intervention. In this case the patient has had over three years of complete comfort and business activities.

CASE 8.—*Summary: A man, 29 years of age, with a fibroma of the acoustic nerve, accurately localized before operation, subsequently exposed and removed; recovery.*

History.—L. A. S., aged 29 years, was referred to the neurosurgical service by Dr. William G. Spiller with the following history:

Three years ago the patient noticed that he did not hear well with his right ear; and ten months ago his hearing, he observed, had become much worse. Seventeen months ago, when he became tired and nervous he began to have a "roaring" in the right ear. He noticed a peculiar sensation in his gums and cheek on the right side, twenty months ago and fourteen months ago difficulty in writing and shaving. Nothing of consequence developed for almost a year when (five months ago) he began to have trouble in walking, with a tendency to stagger, usually to the right, when walking in the dark or on a narrow path. A month later he first complained of disturbance of vision; he could not focus accurately, particularly when moving about. Objects looked blurred, but there was no dizziness. Now, when reaching for an object his hand miscalculates the distance by an inch or so and in going upstairs he has to concentrate on the movements of his right foot.

The chronologic sequence of events is: Three years ago, there was disturbance of hearing (right); twenty-two months ago, deafness more pronounced (right); twenty months ago, sensory disturbances of gum and cheek (right); seventeen months ago, tinnitus in right ear; fourteen months ago, awkwardness in movements of right hand; four months ago, staggering in walking; three months ago, visual disturbances; at present, awkwardness in movements of the right hand and right foot.

Physical Examination.—Cranial Nerves: The first, third, fourth, sixth, tenth and eleventh nerves were normal. There were no ocular palsies. Nystagmus was present when looking to the right or upward (ophthalmic report). Fifth Nerve: The sensation in his face differed on the two sides. When he was pricked with a pin in the distribution of the fifth nerve he felt it as a sharp prick on the left side, but on the right side he said it felt "muffled," though he knew it was sharp, not dull. The sensation was the same on the two sides in the cervical distribution in the lower part of the face. When he chewed he used the left side because he said the right cheek and gums felt as if they had been burned. The right eyeball and cornea when touched with the head of a pin showed no contraction of the lids; the left conjunctival reflex was normal. When he bit, there was no difference in the masseter or temporal contractions on the two sides.

Seventh Nerve: When he wrinkled his forehead the lines were not quite as deep at the extreme right as on the left side, and there was a gradual lowering of the eyebrow on that side so that in order to keep his forehead

wrinkled he had to make repeated efforts, but this was slight. When he showed his teeth both corners of his mouth were drawn back equally, but soon the right side began to relax, and there was a twitching of the muscles in an effort to keep the corner drawn up. The resistance to raising the upper lid when the eye was closed was slightly less in the right eye (Dr. B. A. Randall).

Eighth Nerve: The right ear seemed deaf for all lower tones and even the higher ones were less well heard than on the left. The left ear responded normally to all tests. Past pointing was not spontaneously present, but easily though briefly elicited by rotation or touch. There would seem to be some brainstem lesion anterior to the cerebellar peduncles. The absence of ocular evidence suggested a very small lesion. He could not hear a watch tick close to or even on his right ear. The hearing in his left ear was apparently normal.

Ninth Nerve: The soft palate was normal.

Twelfth Nerve: There was no deviation in the protruded tongue.

Sensation: In the upper extremities, sensation was normal for touch and pain. There was no asterognosis and no loss of sense of position. In the lower extremities there was no loss of sense of position and no disturbance of sensation.

Reflexes: In the upper extremities the reflexes were equal and normal. In the lower extremities the Achilles tendon reflexes were prompt and equal. The right patellar reflex was a trifle prompter than the left. There was no Babinski sign.

Cerebellar Disturbances: There was slight but distinct dysmetria and adiadokokinesis in the right hand. When he wrote he did not slide his right hand along the paper (dyssynergia).

Gait: With his eyes open the gait and station were normal; with eyes closed, the station was good but in walking there was a tendency toward deviation to one side or the other.

Roentgenogram: There was no evidence of any sella deformation. In all respects negative.

The Wassermann reaction of the blood was a doubtful negative. The cerebrospinal fluid was negative.

Résumé of Symptoms: The symptoms were: deafness in the right ear, tinnitus in the right ear, hypesthesia in the right trigeminal zone, facial paralysis partial (right), retinal veins a little larger than normal, dysmetria of the right upper extremity, dyssynergia of the right upper extremity, adiadokokinesis of the right upper extremity, patella reflex a trifle prompter on the right side and deviation to the right in walking.

Preoperative Diagnosis: Acoustic tumor.

Operation.—Ether anesthesia was used, endopharyngeal method. A suboccipital craniectomy was performed with extirpation of the tumor. The bone was removed from over both cerebellar hemispheres, the occipital sinus ligated and the dural flap reflected. There was an increased intracranial pressure, but when the fluid in the basal cistern escaped, the pressure was restored to within normal limits. Displacement of the right cerebellar hemisphere disclosed an acoustic tumor about the size of a grape. The tumor was bisected and removed in segments. Hemorrhage from two points was controlled with muscle grafts. Closure was made without drainage.

At the conclusion of the operation 600 c.c. of citrated blood was transfused.

Postoperative Course.—The patient's convalescence was somewhat prolonged. He was extremely apprehensive throughout. For a long while he complained

of difficulty in swallowing and would not take solid food, although he never choked when taking liquid food as one observes when there is any disturbance of the muscles of deglutition. He complained also for some time of mucus collecting in the throat. But apart from these symptoms there were no unusual complications.

Pathologic Report.—The tumor consisted of spindle-shaped cells having a parallel arrangement and forming bundles. The tumor contained a fair amount of connective tissue and some round nuclei, which were perhaps in part at least transverse sections of spindle-shaped nuclei. The tumor was a typical fibroma (Dr. W. G. Spiller), the characteristic tumor of the acoustic nerve.

Comment.—The sequence of events in the early history were characteristic of an acoustic tumor. Of significance in the diagnosis was the initial deafness. Three years before the operation the patient noticed that he was becoming deaf and for many months this was the only symptom. Then followed in sequence the involvement of the sensory root (hypesthesia), tinnitus and the involvement of the facial nerve. After that there were evidences of disturbance of cerebellar function, dysmetria, dyssynergia, adiadokokinesis and staggering. The primal disturbance of hearing, antedating by so many months any other symptom, either of the cranial nerves or of the cerebellum, was of great significance in the differentiation of angle tumors, other than those originating in the acoustic nerve or of tumors of the cerebellum with secondary invasion of the cranial nerves.

Up to the time of operation there had been no obstruction to the outlet of the ventricles, hence the absence of pressure phenomena. Apart from a slight fulness of the retinal veins there were none. The absence of ventricular distention was observed at the operation, since once the fluid in the posterior fossa escaped there was no evidence of pressure, and it was not necessary, therefore, as in many explorations, to evacuate the ventricles. The right hemisphere was displaced without any difficulty sufficiently for the manipulations necessary for the removal of the tumor.

The tumor, as reported, was a fibroma and not as in many instances an endothelioma. But even though it was a fibroma, there is a possibility of recurrence. These two tumors, fibroma and endothelioma, have similar characteristics; each is relatively benign, but each may recur. An endothelioma may recur as a diffuse process and a fibroma may become readily a spindle cell sarcoma (Spiller).

The surgical problem was devoid of any technical difficulties. The accuracy of localization and the character of the tumor rendered the exposure of the growth and its removal a relatively simple procedure.

The resort to blood transfusion after prolonged intracranial procedures, particularly after suboccipital operations, we believe has merit.

It has not been our practice to reserve transfusions for those cases in which hemorrhage has been profuse. This in itself constitutes a positive indication. But when there has been a prolonged anesthesia and a prolonged operative siege we find that the reaction from these after transfusion is prompter, and the convalescence shortened. From the condition of the patient the day after the operation, when transfusion has been practiced, one would not suspect that he had been subjected to a protracted ordeal on the operating table the day before. The value of blood transfusion in this particular field is enthusiastically recognized by all who come in contact with the patient.

ABSTRACT OF DISCUSSION

Dr. Charles K. Mills of Philadelphia said that, with regard to very small growths, he had had several experiences, one of the growths being in Broca's area and another in the parietal region. The paper was instructive in that it supported the older so-called classic view of aphasia as against the hypotheses of Marie. With regard to epilepsia continua, Dr. Mills said that he had had several cases, for one of which he had had a cranial-cerebral operation over the motor area performed by Dr. Frazier. No lesion was revealed. Dr. Mills said he recalled that Dr. Burr had, some years ago, reported a case of epilepsia continua and discussed the subject in a paper. Of course the whole subject belongs to the old and interesting field of accurate cerebral localization.

Dr. Mills remembered quite clearly the case to which Dr. Spiller alluded, in which they had made the diagnosis of a lesion in the facial cortical center, necropsy showing that the lesion was in the cerebellopontile angle. Recurring facial spasm of moderate intensity was a striking feature in this case. The spreading of jacksonian limb symptoms in these cases of cerebellar pontile lesions was, Dr. Mills thought, not difficult to explain, owing to the proximity of the pyramidal tracts.

Dr. H. H. Hoppe of Cincinnati said, in connection with the small tumors of the psychomotor area, that it might be interesting to know that at times they are tubercles, which are arrested in their growth, become calcified and may remain latent for many years.

A patient was brought into the Cincinnati General Hospital with a supposed stroke of apoplexy on the right side, sudden in onset. He died without regaining consciousness. Necropsy revealed a calcified mass, about the size and shape of a peanut, just below the cortex of the rolandic area in the ascending frontal convolution on the left side. The history of the case, obtained from a relative, disclosed that forty years before, the patient, when 16 years of age, had developed gradually, in the course of several months, a right-sided weakness of the body. He recovered from this and for forty years afterward did hard manual labor.

The pathologic examination revealed that the mass was a calcified tubercle surrounded by a cartilaginous envelope.

Dr. Hugh T. Patrick of Chicago mentioned a case of epilepsia partialis continua, in a boy of about 10 years. The first symptom had been intermittent shaking of the right foot. In other words, it continued uninterruptedly for a while and then stopped for a time. The movement very much resembled that of some of the shakers with war neuroses. Another feature of it was that it

continued during sleep. When Dr. Patrick saw the boy he was sound asleep but the right leg was in this continuous clonic spasm. By that time the boy had also developed typical jacksonian fits, beginning in the right leg. The shaking was interrupted by a more tonic spasm, succeeded by a clonic spasm that spread over the entire right side. It was a case of cortical tumor of the left hemisphere.

Dr. William G. Spiller of Philadelphia said he was aware of the paper written by Dr. Burr, which was published some years after the one by Dr. Martin and himself, and a reference was made in the paper by Dr. Frazier and himself to Dr. Burr's paper.

ACUTE ASCENDING MYELITIS OF THE INFECTIOUS TYPE

ITS POSSIBLE RELATIONSHIP TO POLIOMYELITIS IN A CERTAIN GROUP OF CASES *

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The experimental and clinical work of Homen, Marinesco, T. Buzzard, Russel, Strumpell, Gowers and many others has been of importance in placing the clinical picture of acute disseminated myelitis on its correct pathologic and etiologic basis. It is not the object of this paper to discuss, from either the pathologic or the clinical standpoint, acute disseminated myelitis as such, but to study acute ascending myelitis, an anatomic variation of the same disease. As a means of comparison we have chosen a case of acute ascending poliomyelitis presenting both typical clinical manifestations and pathologic evidence of this disease. Both cases occurred on the service of Dr. F. W. Peabody, in the medical wards of the Peter Bent Brigham Hospital, and with his consent we summarize the cases.

REPORT OF CASES

CASE 1.—*History.*—F. T. C., woman, aged 28, an American housewife, who complained of stiffness of the neck, gave a negative family and marital history. There had been no pregnancies. The patient was moderate in her diet and had a fair appetite. The bowels were regular. She had had chicken pox, mumps and measles in childhood. At 14 years she was in bed three months with a complaint of general weakness, diagnosed tuberculosis of the right lung apex. Later she was informed that the process was arrested. Six years before she came under our observation she had had an attack of bilateral otitis media, but had recovered without suppuration or deafness. There had been frequent attacks of tonsillitis in the last four or five years. There was no history of syphilitic infection. Menstruation began at the age of 16. The periods were irregular, coming at intervals of three, four or five months, lasting two weeks and associated with considerable pain. The patient's weight had been 114 pounds two years before; her present weight is 98 pounds. Three weeks previously she had contracted rhinitis, characterized by sneezing, nasal discharge and subjective fever. Six days previously, July 31, 1920, she had developed pain in the pit of the stomach and the back. The following day

* From the medical clinic and pathologic laboratory of the Peter Bent Brigham Hospital, Boston, Mass.

four teeth were extracted. On August 2, she complained of neck soreness, and the following day she developed fever and stiffness of the neck muscles, with headache and insomnia. The next morning there was pain localized in the back and retention of urine. The day previous to admission she vomited several times and her neck became painful to flexion, retention of urine continued and both legs became paralyzed rather suddenly.

Physical Examination.—The patient was a thin, sick appearing woman, mentally clear, lying in bed with head somewhat hyperextended. She was cooperative, but was apparently suffering considerable pain. Her apparent weakness was augmented by the pain on movement of head and body. The skull was symmetrical; the scalp clean. The pupils were regular and equal, and reacted to light and in accommodation. There were no external ocular palsies. The sclerae and conjunctivae were normal. There was no nystagmus. The optic nerve heads and retinas showed no abnormalities. The hearing was equally good in both ears. There was no discharge from the meatus; and no obstruction or discharge from either nostril. The lips were of good color and showed no herpes. There was no disturbance of the muscles of facial expression. The teeth were in fair condition. Several upper and lower teeth had been recently extracted but reaction was slight around the gums. The tongue protruded in the midline without tremor. The tonsils were not enlarged. The palate contracted symmetrically and the gag reflex was present. The voice had not changed in quality and no difficulty was experienced in swallowing. The neck was somewhat extended. Turning of the chin to the side caused pain.

There were no athetoid or choreiform movements of the arms. Fibrillary tremors were not noted in either arm, and there was no atrophy. Strength was equal in both arms and hands, and there was no dysmetria. All forms of sensation were normal. Biceps, triceps and radial periosteal reflexes were neither diminished nor exaggerated, and were equal. There was a flaccid paralysis of both legs save for a slight abduction movement at the hips. The movements of both feet were impaired. Neither leg could be raised from the bed; flexion at the knees was weak but extension at the knee was absent. Both legs showed fibrillary tremors. They lay absolutely flaccid and both knee jerks were absent. The tibialis anticus and the peroneal muscular reflexes were absent on both sides. The Achilles reflexes were hyperactive. There was no Babinski sign, and all forms of sensation were unimpaired. A bilateral Kernig's sign of slight degree was present.

Course of the Disease.—The following day all the voluntary movements of the lower limbs were abolished and the legs lay absolutely flaccid. The Achilles jerks were absent; sensation was retained; and Kernig's sign was lost. Catheterization for relief of retention was necessary. During the course of the day the intercostal muscles ceased to act and the abdominal reflexes were absent. All the trunk muscles were paralyzed and breathing was purely diaphragmatic. The arms were held in a flexed position. On the afternoon of the same day, the left arm assumed an extended position with loss of voluntary motion and abolition of the deep reflexes. Shortly afterward the right arm was in an identical position and similarly showed signs of a flaccid palsy. There were no sensory disturbances. All deep reflexes were absent.

On admission the temperature was 101.5 F., pulse 104, respirations twenty per minute. On the following day (day of death) her temperature was 101 F., pulse 100, respirations 30, with a terminal rise of temperature to 102.4 F., a stationary pulse rate and respirations 40.

Clinical Pathology.—The urine (catheterized specimen) was amber; alkaline; with a specific gravity of 1.022; albumin, the slightest possible trace; sugar, none; sediment, no casts, and no red or white blood cells.

The blood examination revealed hemoglobin 90; white cells 12,800; 85 per cent. polymorphonuclears; 15 per cent. mononuclear leukocytes.

The cerebrospinal fluid, on admission, showed 246 cells, 95 per cent of which were lymphocytes, 5 per cent. polymorphonuclear leukocytes; globulin ++. Fehling's solution was reduced. There were no bacteria seen in the smear. Cultures were made and remained sterile.

A fibrin web[1] formed over night. No acid-fast organisms were seen in stained smears. The blood Wassermann reaction was negative.

Outcome.—The case, then, was one of a young woman mentally clear and alert, absolutely paralyzed from the neck caudad. The accessory muscles of respiration were prominent and overacting. The sternomastoids, alae nasi and the diaphragm all showed strikingly their increased responsibility. The patient realized her distress. She requested that the covers be removed from her abdomen and spoke only in monosyllables. The respirations became irregular and the patient died that evening of bilateral diaphragmatic involvement. There were no cranial nerve palsies as far as could be determined. Her mental status was unimpaired to the end. The duration of the illness was six days.

Necropsy Findings.—Necropsy was performed twelve hours postmortem. The diagnosis was acute anterior poliomyelitis, acute splenitis, and healed pulmonary tuberculosis (right apex).

The body was that of a well developed, fairly well nourished woman. The left axillary gland was a trifle enlarged. The mesenteric lymph nodes were enlarged, moist and reddened on section. Examination of the heart revealed numerous small, red, petechial hemorrhages beneath the pericardium. Otherwise it was not remarkable. The valves and endocardium were normal; the myocardium appeared slightly paler than normal. The lungs were negative except for congestion of the dependent parts. They had a normal pink color, a "downy" crepitant feel and were voluminous in the anterior portions. At the apex of the right lung was a small, opaque, depressed scar. On cutting, the surface of the dependent parts was smooth, and by pressure a large amount of dark, reddish fluid was expressed. The bronchial lymph nodes were swollen and congested. The spleen weighed 130 gm. The cut surface was dark red and of a somewhat soft consistence. Examination of the gastro-intestinal tract indicated that the stomach and large and small intestines were evidently normal, with the exception of a few Peyer's patches which were congested. The weight of the liver was 1,315 gm. It appeared to be normal. The kidneys and suprarenal glands were normal.

In examination of the skull, on removing the calvarium, the dura appeared normal and no fluid escaped. The sinuses contained a few small red clots but no thrombi. The middle ears were normal. Examination of the brain revealed a moderate degree of injection of the pial vessels and a slight diffuse reddening of the gray and white matter throughout. It was not, however, a wet brain. Sections of the cerebrum, cerebellum, medulla, pons and basal ganglions showed no definite lesion. Examination of the spinal cord revealed the superficial veins much distended and the whole cord, particularly in the lumbar and thoracic regions, swollen. Sections taken from various

1. Fibrin web formation in poliomyelitis in spinal fluid standing over night as mentioned by Dubois, Am. J. Obst. **74**:1074, 1916, is not an unusual event.

levels showed in the lumbar cord marked softening of the gray matter, particularly of the anterior horns, so that they bulged from the cut surface, in some places being hemorrhagic and so soft that a cavity was easily formed by slightly scraping the surface with a knife.

The vessels of the white matter were injected but no definite lesion was discerned there. The degeneration of the anterior horns was marked in the lumbar and lower thoracic regions, but became less in ascending, until, in the cervical segments, only a diffuse reddening and some swelling of the cord was noted. No increased amount of spinal fluid was apparent. A frozen section of the lumbar cord revealed almost entire replacement of the anterior horns by large endothelial (?) cells and polymorphonuclears, very marked vascular dilatation and perivascular mononuclear infiltration. An emulsion of cervical cord in physiologic sodium chlorid solution was made and 5 c.c. injected into the peritoneal cavity of a rabbit. No signs of a reaction of any kind developed during the ensuing four weeks.

Microscopic Examination.—The heart, lungs, kidneys, pancreas and suprarenal glands revealed nothing of importance. In the spleen the pulp sinuses were markedly congested and the malpighian bodies were very small. There was marked hydrops of the liver cells and in the center of the vacuoles there were frequently a few colloid droplets, often the center of a radiating fibrin meshwork. Necrosis of a single liver cell with invasion by polymorphonuclears was not uncommon.

A section of the colon revealed moderate congestion of the mucosal vessels and infiltration of mucosa by mononuclear cells and eosinophils in numbers. Some of these mononuclear cells were endothelial leukocytes filled with hemosiderin pigment. The solitary follicles were hyperplastic, mitotic figures being abundant. Many endothelial cells in mitosis were also present in these areas.

Lymph nodes from the mesentery revealed marked lymph stasis. There were a few small germ centers but as a whole they were atrophic. Most of the cells were small lymphocytes.

Examination of sections from the frontal, temporal, parietal and occipital lobes of the brain revealed congestion of the blood vessels and edema of the perivascular sheaths. In some sections also a moderate number of polymorphonuclear cells were present, diffusely scattered in the white matter, and rarely one was seen attacking a spider cell, the nucleus of which had undergone chromatolysis. The cerebellum was normal. Sections from the level of the fifth, sixth, seventh and eighth cranial nerves showed extensive lesions of the nerve cells and blood vessels. No nuclei had entirely escaped damage and the lesions varied from loss of cytoplasmic granulation and pyknosis of nuclei, to necrosis with phagocytosis by polymorphonuclear neutrophils and endothelial cells. Minute hemorrhages from congested capillaries were found here and there in nuclei and pyramidal fiber tracts. There were also perivascular edema and infiltration about the blood vessels, both in the cord and in the meninges by polymorphonuclears and lymphocytes. More marked lesions were present in the medulla than in the pons. Few nerve cells were intact and more of these revealed degenerative changes in nucleus and cytoplasm. The gray matter was disorganized by the tremendous invasion of polymorphonuclear leukocytes and large neuronophages which had replaced degenerated cells in the nucleus gracilis, radicis spinalis trigemini, and the dorsal nucleus of the tenth nerve. The white matter also manifested some change, the axons of many nerve fibers in funiculi gracilis, cuneatus and fasiculus anterior proprius having disappeared and vacuoles being left in their places. The blood vessels of the meninges, includ-

ing those of the anterior and posterior median fissures, were dilated and surrounded by polymorphonuclears and endothelial leukocytes in large numbers. There was no dilatation of the canalis centralis. Proceeding cephalad in the medulla the lesions were well marked but the olivary nucleus manifested less involvement than the other nuclei. The damage was always symmetrical and there was little to choose between the various levels as to which indicated the older lesion, but the higher levels were undoubtedly less affected than the lower. No reparative changes were seen except the phagocytosis of necrotic nerve cells.

Examination of the spinal cord revealed striking and extensive lesions in the gray matter at all levels. It was difficult to say where the earliest lesion had been. In the sacral cord it was as marked as in the lumbar with slightly less horn involvement in the mid-dorsal region than elsewhere. There was congestion of the vessels and edema of the meninges and cord with many recent hemorrhages into the gray and white matter. An intact ganglion cell was rare. Necrosis of ganglion cells and neuronophagia was conspicuous at all levels with infiltration of anterior and posterior horns by endothelial cells and polymorphonuclear neutrophils. Perivascular mononuclear infiltration was to be seen about practically every vessel in the meninges and cord. There was acute degeneration of fibers in the lateral, posterior and anterior tracts, although this damage to the white matter was less in the mid-dorsal level than above or below it. There was also edema and mononuclear infiltration of the afferent nerve roots. No organisms were found by Goodpasture's method.

This was a typical case of rapidly ascending acute anterior poliomyelitis.

Case 2.—*History.*—G. S. F., aged 39, American, a salesman, complained of "pain in the back and paralysis of the legs."[2] The family history was negative. The patient had been married five years; his wife was healthy, and she had had one healthy child and one miscarriage. The patient had never drunk to excess. His appetite was good; the bowels regular. He had had measles, mumps and diphtheria as a child, and an arthritic attack four years before. This was not typical of rheumatic fever, however, and there were no sequelae. He had had severe tonsillitis and laryngitis two years before. No venereal history was obtained. He had suffered a fracture of the left leg and subsequent bone plate repair three years before.

There had been no loss of weight up to the present illness. This had begun acutely Monday, June 1, 1920, with severe headache and pain in the lower part of the back. The same evening he became somewhat delirious with a temperature of 101.5 F. The headache continued the next day. The patient was clear mentally. A doctor was called who suspected meningeal involvement. Tuesday night the patient was again delirious with a temperature of 102 F. He vomited without nausea. Wednesday morning he was entirely helpless from the hips distally, save for slight movement of the right leg. Urine was retained, necessitating catheterization. The headache continued and the patient was at times delirious. He was admitted to the hospital the same day.

Physical Examination.—This revealed a well developed and well-nourished white man, mentally somewhat clouded, assuming by preference a prone position. The skull was symmetrical and showed no tenderness. The scalp was clean. There were no scars. The facies suggested a febrile condition. There were no seventh nerve palsies. The pupils reacted rather sluggishly to light but were regular, equal and reacted in accommodation. There were no external ocular palsies. There was slight ptosis of the left lid, but no exophthalmos.

2. The history was obtained from the wife and the sister.

There was some sclerosis of the retinal vessels and a definite blurring of the lower part of the left disk. The ears revealed no deafness to superficial tests. There were no signs of motor fifth nerve involvement. The lips were of good color. The teeth were in fair condition with a few cavities noticeable. The tonsils were red but not enlarged and showed no exudate. There were no mucous patches in the mouth. The palate was symmetrical and the gag reflex was present. The tongue protruded in the midline without tremor. The voice was soft; no hoarseness, dysarthria or nasal element was noted. Examination of the vocal cords was not attempted. The neck was slightly stiff but the movements were unimpaired. There was slight cervical adenopathy in the supraclavicular region.

The thorax was symmetrical. The chest expanded but little on inspiration, the breathing being diaphragmatic. The heart was not enlarged but there was a slight increase in retromanubrial dulness. No thrills were felt. There was a protodiastolic gallop rhythm at the apex and at the pulmonic area a snapping second sound. A loud systolic murmur was heard over the entire precordia with its greatest intensity over the pulmonic area. No diastolic murmurs were heard. The radial pulses were equal and synchronous, the rate rapid and the pulse not well sustained. The walls were palpable. The blood pressure was: systolic, 175; diastolic, 30. Examination of the lungs revealed a slight relative dulness over the left back with prolonged expiration but no râles. The right lung was negative. The abdomen was protuberant but symmetrical. No abnormal tenderness, spasm or masses were noted. Respiratory movements were entirely abdominal in character. The abdominal reflexes were not obtained.

The arms were held in the flexed position and could not be raised above the head. The muscular power in the hands and arms was impaired. Dysmetria was noted in the performance of movements of precision. There were no involuntary movements or fibrillary tremors. There was no wasting. Faradic and galvanic tests were not attempted. There was some loss of muscle tone. The legs revealed paralysis of the absolutely flaccid type. Passive movement was in no way resisted, gravity determining their position when support was removed. There were no involuntary movements, fibrillary tremors or atrophy. The knee jerks and Achilles' tendon reflexes were abolished. Kernig's sign was absent.

The sensations of pain, temperature and touch were markedly impaired from the level of the sixth rib distally. Over the left leg there was a slight appreciation of tactile sense but this was indefinite. Deep sensibility was impaired in both extremities.

The patient did not complain of pain.

Course of the Disease.—On the following day, June 4, paralysis had ascended as indicated by the following note: "Patient is semicomatose, irrational and unable to cooperate. Breathing is rapid and diaphragmatic in type. There is flaccid paralysis of both arms and legs." It is unfortunate that no note was made concerning the muscles of the neck.

In the afternoon, the patient's respirations were more shallow, and bulbar symptoms developed. Speech became drawling in character, the tongue was protruded with difficulty and swallowing caused regurgitation into the nose, and choking. Apparently rational and irrational periods alternated until the patient sank into coma from which he did not arouse. Death occurred the morning of the third day from respiratory failure. The duration of the disease was five days. The temperature on admission had been 103 F., pulse rate 100, respirations twenty-three per minute. On the following day the temperature

was 106 F., with a relatively slow pulse rate of 102, respirations 26. There was a terminal rise of temperature the morning of death, reaching 109 F. per rectum, pulse 106, respirations 15, becoming irregular. The chart thus showed an involvement of the vital centers, i.e., extreme temperature, rapid pulse and slow, irregular respirations.

Clinical Pathology.—The urine (catheterized specimen) on admission was amber; acid; specific gravity 1.020; no albumin; no sugar; sediment, many white blood cells, numerous spermatozoa, and a few hyaline casts.

The urine (examined on the following day) was orange yellow; alkaline; specific gravity 1.022; no albumin; no sugar; a few white blood cells, no red blood cells, and no casts.

The examination of the blood (on admission) revealed hemoglobin 90 per cent., white blood cells 15,000, 80 per cent. polymorphonuclears, 16 per cent. lymphocytes, and 4 per cent. mononuclear cells.

The spinal fluid (on admission 30 c.c. fluid removed) contained 387 cells per cubic millimeter, 60 per cent. of which were polymorphonuclear, 40 per cent. mononuclear cells; globulin +. No organisms were seen in the stained specimens.

Cultures were made and the fluid was injected into the peritoneal cavity of a guinea-pig.

On the following day, a second lumbar puncture was made. Twenty c.c. of clear fluid was removed, showing 250 cells per cubic millimeter, 75 per cent. polymorphonuclear cells; globulin + +.

Cultures from the fluid of the previous day showed no growth. No fibrin web was formed in the specimen on standing.

A guinea-pig killed six weeks after inoculation had no evidence of tuberculosis.

The blood culture (on admission) gave no growth on blood serum and dextrose broth during ten days incubation.

The blood Wassermann reaction was negative; the spinal fluid Wassermann reaction was negative with 2 c.c.

Necropsy Findings.—Necropsy was performed eight and one half hours post-mortem. The diagnoses were acute diffuse ascending myelitis, bronchopneumonia, and passive congestion of the viscera.

The body was that of a well-nourished and well-developed white adult male. Eyes, ears and nasal passages were negative. The teeth were in fair condition. There were two small calcified lymph nodes in the mesentery. The diaphragm extended to normal limits on both sides. The pleural and pericardial cavity and mediastinum were normal. The heart was normal. The lower lobes of both lungs revealed small patchy areas of bronchopneumonia and much edema. The weight of the spleen was 120 gm. It was of normal size; externally, it was not remarkable. On section, it was of rather beefy color. The pulp was quite soft and somewhat diffluent. The gastro-intestinal tract was normal, as was the pancreas. The liver, which weighed 1,800 gm., was much congested. The kidneys were normal except for moderate congestion. The suprarenal glands were normal.

The dura was normal. The brain weighed 1,750 gm. It was unusually soft and edematous, and there was some flattening of the convolutions. The surface was everywhere injected, particularly over the cerebral hemispheres. On section the cortex was gray and slightly suffused with red. Section through the pons revealed a small hemorrhage just below the lower cerebral peduncles. There was marked injection of the decussation of the pyramids. Coronal sec-

tion through the brain in the region of the internal capsules revealed nothing remarkable. The venous sinuses contained no thrombi. The middle ears and nasopharynx appeared normal.

Examination of the spinal cord revealed no evidence of increased fluid or clouding of the fluid. The meninges were injected throughout the entire length of the cord. The upper dorsal and cervical regions were soft, edematous and rather doughy. A transverse section revealed the normal markings almost completely obliterated, and distinction between gray and white matter in these regions was difficult, and in places impossible. At the lumbar level, there was softening, but less than higher up, and the markings were fairly well defined. The cord everywhere was congested.

Microscopic Examination.—The lungs, spleen and kidneys revealed nothing of importance.

The spleen showed moderate congestion. The lymphoid centers were small.

Examination of the liver revealed a few focal necroses with invasion by endothelial phagocytes.

The frontal, parietal, temporal and occipital lobes of the brain showed vascular congestion and edema of the cortex and meninges. A moderate number of polymorphonuclear wandering cells were present in the pyramidal zones and rarely a glia cell in mitosis was seen. No degeneration of nerve fibers was shown by Marchi's method.

More marked congestion and edema were observed in the cerebellum than in the cerebrum. An occasional ganglion cell of the nucleus dentatus had undergone degeneration as shown by retraction of the axon, chromatolysis of nucleus and loss of cytoplasmic granulation. No neuronophagia, however, was seen nor any Marchi degeneration.

Examination of the pons in addition to much edema and congestion, showed a few small, recent hemorrhages, found at many levels. Scattered rather diffusely through the tissue were nongranular polymorphonuclear cells and many cells of this same type with mitotic figures. Marchi's method revealed acute degeneration of varying amount in a few fibers of the pyramidal tracts and in a small number of the spinal roots of the fifth nerve and median fillet. Perivascular mononuclear infiltration was present in the gray matter and the pia. No neuronophagia had occurred although now and then a ganglion cell had lost its cytoplasmic granules and dendrites.

The same lesions found in the pons were present but were more widespread and pronounced in the medulla. Beneath the floor of the fourth ventricle, proliferation of neuroglia cells and degeneration of the nerve cells of the nucleus ambiguous had occurred, and a few necrotic ganglion cells were invaded by phagocytes. Most of the blood vessels were surrounded by collars of small round cells. Proceeding caudad the lesions in the gray matter became less pronounced with the white matter showing more minute hemorrhages and acute degeneration of myelin sheaths and axons.

The most marked lesions of the spinal cord were in the white matter. Acute degeneration of the myelin sheaths and axons had occurred at all levels but was more marked in the cervical and lumbar levels. This was brought out strikingly by Marchi's method, but was also noticeable with eosin and methylene blue staining. There was no predilection for any special tracts, the fibers in the posterior, lateral and anterior pyramidal tracts being involved. No reparative gliosis had taken place. The sacral and thoracic regions disclosed slightly less myelin degeneration. At all levels was found a perivascular mononuclear reaction in both gray and white matter and in the pia mater. The

anterior horn cells showed comparatively little. There was no actual neuronophagia seen below the medulla, although many cells appeared shrunken and had lost their cytoplasmic granules. Many nuclei were swollen, others were pyknotic or had undergone karyolysis. Nongranular polymorphonuclear cells were present in small numbers in both gray and white matter but did not appear to be active in phagocytosis. There were minute recent hemorrhages in the white matter in the lateral pyramidal tracts, in the lateral ground bundles of the cervical cord and in corresponding areas in the lumbar cord. A few hemorrhages were also present in Clark's columns and in the anterior cornua. In the upper cervical cord slight dilatation of the canalis centralis had occurred.

The dorsal and ventral radices disclosed very little cellular reaction and this only beneath the epineurium. But acute degeneration of many fibers was seen in Marchi preparations and occurred in both dorsal and ventral roots in equal amounts. At the cervical level some fibers had completely degenerated; others revealed slight changes. About half seemed to have escaped altogether. No ganglions were available for study. No organisms could be demonstrated in sections of the cord from various levels stained with methylene blue, by Goodpasture's or by Levaditi's method. The lesions in the cord were, as a whole, quite marked at all levels. The sacral segments had suffered as much as the lumbar and it was difficult to determine which was affected first. The thoracic and cervical levels, however, were less involved than the lumbar and sacral.

DISCUSSION OF THE TWO CASES

For comparison we have presented two cases, clinically similar in some respects, unlike in others.

Case 1 approaches the classic picture of acute poliomyelitis of the ascending type. It is that of a young woman, who, apparently in good health, was attacked more or less suddenly with pain in the back, followed by soreness and stiffness of the neck, and by headache and insomnia. Subsequently there developed vomiting, retention of urine and a flaccid paralysis of the lower extremities without sensory disturbances. Anatomically, the virus probably caused early and extensive destruction in the gray matter of the lumbar region, subsequently ascending and descending; for it will be recalled that the knee jerks were absent on admission, as were also the tibialis muscle responses, while the Achilles jerks were hyperactive, only to be lost the following day in conjunction with a complete paralysis of the small muscles of the foot, the muscles of the calf, and the ham string muscles. We are thus correct in assuming an extension of the process, at least to the second sacral segment.

There is abundant evidence of an ascent of the process in the anterior horns. On the day following admission, the abdominal reflexes were abolished and the trunk and intercostal muscles were paralyzed, with consequent diaphragmatic breathing. The arms, as stated, were held in flexion. Theoretically, at least, this sign might be interpreted as a measure of the muscle power of the flexors versus the extensors, the latter being the weaker. Segmentally, one would expect, in an

ascending palsy, that the motor cells of the seventh and sixth cervical ventral roots would be attacked at a slightly earlier stage than the motor cells of the sixth and fifth cervical radices. The presence of equally active triceps and biceps reflexes, however, makes difficult such a nice differentiation.

After some hours, both arms became completely paralyzed with loss of tendon reflexes. The signs indicate, therefore, that the lesion had reached the fifth cervical segment with remarkable rapidity. From this stage until the end, it was a matter of but a few hours, the virus attacking the fourth segment, possibly the third, and death resulting from bilateral phrenic nerve palsy.

Her struggle for life was classical and follows so closely a description given by Peabody, Draper and Dochez [3] that we might as well quote from their monograph: "Instead of the normal smooth rhythmic cycle there may be sharp forcible movements of the abdominal wall on expiration. Pressure over the abdomen causes distress and interference with breathing. The alae nasi dilate with inspiration and the accessory muscles of respiration in the neck begin to come into play. As the diaphragm weakens the neck muscles become more and more prominent until it seems as if the whole work of breathing depended on them. The sternomastoids pull on the clavicle and in one case they caused a partial dislocation of the sternoclavicular joint with each inspiratory effort. The head is thrown back and with every breath the lower jaw is pushed forward and downward in a gasping attempt to get air." Such was the terminal picture of Case 1.

The fibrin web formation in the spinal fluid is exceptional but does occur in poliomyelitis.[1] The fluid otherwise is in accord with the meningeal involvement of poliomyelitis, a constant accompaniment of the disease.

The pathologic picture confirms the clinical diagnosis.

Our purpose is not to discuss acute ascending poliomyelitis, the symptoms and pathology of which are known and for the most part well recognized, but as previously stated, to study Case 2, one of acute ascending myelitis, and to offer an analogy between them, perhaps so far as to suggest the same etiology in a small group with this disease complex. This is not an original idea. Recently there have been a few cases of transverse myelitis reported as poliomyelitis of the transverse type. We shall refer to this question later. Suffice it to say, however, that acute myelitis, ascending and descending, is ordinarily classified by most authors as infectious, toxi-infectious, or toxic in

3. Peabody, Draper and Dochez: Monographs of the Rockefeller Institute for Medical Research **70**:98, No. 4, 1912.

origin and that the streptococcus, staphylococcus, pneumococcus, anthrax bacillus, typhoid bacillus, etc., have been held, and rightly so, as the causative micro-organisms.

A clear and descriptive picture of this complex is quoted from F. Buzzard:[4]

> Acute Ascending Myelitis: This forms a morbid entity which can readily be recognized during life. It is characterized by a progressive paralysis of motion and sensation, beginning in the lower extremities and more or less rapidly climbing up the body, segment by segment, until involvement of the respiratory musculature threatens life and not infrequently produces a rapid termination. The extension of the disease to the brain stem is occasionally indicated by the implication of the cranial nerves before death, and the occurrence of optic neuritis has been recorded in nonfatal cases where all other symptoms have been referable to the spinal cord. With the more usual employment of the lumbar puncture the bacteriology is likely to become better known.

The same author states in his Goulstonian Lecture:

> It is a matter of some difficulty to choose between the terms acute ascending myelitis and acute disseminated myelitis, because the one is probably better supported from the clinical, and the other more appropriate from the pathological point of view, but I choose to include the word "ascending" for the reason that it graphically suggests the prominent characteristics of the disease. Acute ascending meningo-myelitis would do equally well and is in most cases more correct. Reference to the meningitis is, however, no more necessary here than is reference to the pleurisy which undoubtedly complicates the great majority of cases of pneumonia.

In glancing back over Case 2 the clinical picture corresponds fairly well, and the diagnosis can, therefore, be considered as falling in this group. To recapitulate: A previously strong, healthy male adult was taken acutely sick with severe headache, pain in the lower back, fever and nocturnal delirium. The following day vomiting was an added symptom and the third day found the patient paralyzed from the hips down, save for slight movement in the right leg. There was retention of urine. The same day (day of admission) there was noted, in addition, a slightly stiff neck and paralysis of the muscles of the trunk with a resulting absolute diaphragmatic type of respiration. Further, and of great importance, there was marked impairment of all forms of sensation from the level of the sixth rib caudad. Thus, from our anatomic and physiologic knowledge, one must assume a diffuse lesion of both the gray and white matter of the cord, if we are able to exclude acute polyneuritis. The flaccid paralysis and the absent tendon reflexes imply damage of the anterior horns; the loss of the sense of pain and temperature implies damage in the lateral columns, and the loss of deep

4. Buzzard: Quoted in Osler and McCrae's Modern Medicine **5**:227.

sensibility designates posterior column involvement. Clinically, then, the process is diffuse and the subsequent course proved it, in addition, to be of an ascending type.

The next day both arms were involved in a flaccid paralysis. It is unfortunate that the patient was irrational the last twenty-four hours, as more accurate determinations of sensibility, especially of the arms, could have been made. Death occurred with definite symptoms of a brain stem lesion.

This case has been grouped under the caption, acute infectious myelitis, because of a suspected infectious etiology. It can, under clinical grouping, be designated acute ascending myelitis or more appropriately, acute diffuse ascending myelitis. "Myelitis" is here used in the strict sense—inflammation of the spinal marrow. It is more a matter of the degree of inflammatory reaction whether or not certain pathologic specimens shall be classified as an infectious or toxic myelitis. Infective myelitis is a vague term in the absence of septicemia, pleocytic spinal fluid or demonstratable infectious foci. The vagueness of the term is augmented by the absence of demonstrable pathogenic organisms, by culture or spinal cord preparations. Toxi-infectious myelitis, on the other hand, is inapplicable in the absence of foci or infection or convalescence from acute infectious disease.

SIMILAR CASES IN THE LITERATURE

The authors of various texts agree on an infectious etiology of acute myelitis (Oppenheim,[5] F. Buzzard,[4] Bing[6] and many others). Such has been proved many times, both experimentally and clinically, and the literature contains many authentic cases.

Cushman's case[7] was one of acute ascending myelitis in the course of typhoid fever and he was able to demonstrate *B. typhosus* in the cord. Schiff, on the other hand, reports a case of typhoid fever whose cord symptoms began at the end of the first week of the disease. He was unable to demonstrate organisms in the cord (quoted by Lépine).[8]

Localized infection in distant parts of the body needs consideration here. That at times such a focus will give rise to symptoms referable to the cord is well recognized. Whether or not the organisms themselves intermittently gain access to the blood stream and by that route localize in the nervous tissue, or whether there is a selective action of

5. Oppenheim: A Textbook of Nervous Diseases, trans. by Bruce, Edinburgh, Otto Schulzes & Co. **1**:315, 1911.

6. Bing: Nervous Diseases, trans. by Allen, New York, Rebman Co., 1915, p. 158.

7. Cushman: Verhandl. d. Cong. f. inn. Med., 1886.

8. Lépine: Rev. de méd. **23**:931, 1903.

their toxins on the cord, is at present difficult to conclude. Homen [9] has shown that the cocci tend to disappear quickly from the substance of the spinal cord after they stimulate an inflammatory reaction. Negative cultures then prove nothing.

Recently Olitsky and Kligler [10] have demonstrated an exotoxin or neurotoxin in *Bacillus dysenteriae* Shiga, producing in rabbits, by intravenous injection, lesions in the gray matter of the medulla and cervical cord consisting of necrosis, hemorrhage, neuron degeneration and perivascular mononuclear reaction.

Orr and Rows,[11] instead of injecting organisms or their toxins into the peripheral nerves, placed a celloidin capsule containing a broth culture of the organisms in contact with the nerve. In their first experiments, they used different kinds of bacteria, but later worked with *Staphylococcus aureus* only.

From their pathologic researches, they conclude that "in the spinal and cranial nerves there is an ascending lymph stream to the central nervous system whose main current lies in the spaces of the perineural sheath. Toxins reach the brain and cord by this route and although they spread to some degree in the lymph spaces of the pia arachnoid and so affect structures at a distance from the point of entrance, they pass for the most part in the main stream along the nerve roots into the substance of the central nervous system."

In the examination of their pathologic specimens all the signs of an ascending perineuritis were apparent, extending from the infected focus to the cord which showed all the lesions of a meningo-myelitis. Beside experimental data, these investigators have shown proof by clinical cases and histopathologic preparations.

With these anatomic considerations, given a theoretical case of myelitis which develops subsequent to an ascending infection along the nerves, it seems more logical to expect myelitis dominating the picture, associated with a secondary meningitis, than the reverse.

Some cases of acute myelitis have shown, either by means of spinal fluid or at necropsy, inflammatory reactions of the meninges. It is probably a constant finding in cases due to the localization of bacteria in the substance of the cord. On the other hand, signs of meningeal inflammation may not be so marked in toxic myelitis of bacterial origin. This in contradistinction to experimental evidence seems the only logical assumption when the spinal fluid shows no inflammatory products. As an example of a well-marked localization of organisms in both the cord proper and the spinal meninges, we refer to the case of acute ascending meningo-myelitis with bacteriologic examination, reported by T. Buz-

9. Homen: Arb. a. d. path. Inst. d. Univ. Helsingfors, 1902.
10. Olitsky and Kligler: J. Exper. M. **31**:19 (Jan.) 1920.
11. Orr and Rows: Brain **36**:271, 1913.

zard and Russell.[12] The cord was soft and congested, showing hemorrhages sufficiently large to be obvious to the unaided eye and more in the gray than in the white matter. The pia arachnoid was milky in appearance over the cord, but over the brain it was normal. Microscopically, in some places the destruction was so great that there was little possibility of distinguishing gray and white matter. In some parts there were enormous numbers of leukocytes and little nerve tissue. All the vessels showed diffuse infiltration of small round cells. There was isolated from the exudate on the meninges and the substance of the cord a gram-negative diplococcus.

For means of comparison, we will refer again to the case reported by Hunt.[13] This patient was suffering from otitis media and a mastoid operation was performed for the complicating mastoiditis. Four days later the patient developed the first symptoms of meningo-myelitis. The picture rapidly developed into an acute disseminated myelitis, but the spinal fluid on two occasions was normal. The first puncture was made a month after the symptoms began.

There were two, or perhaps three, possibilities to explain normal spinal fluid in this connection: first, that bacterial toxins were the causative factor in the myelitis without an accompanying meningitis; second, organisms which had localized in the cord quickly died out, a not uncommon phenomenon, as investigated and noted by Homen. (Marinesco[14] has offered this as an explanation in Case 5 of his series), or third, a spinal puncture might have given positive evidences of an inflammatory reaction at an earlier date.

The world war afforded opportunity for the study of ascending neuritis with subsequent myelitis, but the condition was certainly a rarity. We have not been able to find a typical case. Even the clinical conception of ascending neuritis rests on a very doubtful pathologic basis. The cases, reported by Walshe,[15] of local paralysis following superficial injuries do not seem to be in that category.

Paraplegia urinariae is still unexplained. It is conceivable that an ascending neuritis along the peripheral nerves of the bladder with a localized myelitis may be the important factor concerned, as Leyden has assumed. Walker's[16] three cases of acute ascending paralysis of a most malignant nature had a marked cystitis of long duration. No necropsies were obtained.

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12. Buzzard and Russell: Clin. Soc. Trans., London **31**:185, 1898.
13. Hunt: Boston M. & S. J. **174**:788 (June 1) 1916.
14. Marinesco: Nouv. iconog. de la Salpêtrière **13**:561, 1900.
15. Walshe: Brain **42**:338, Pt. 4, 1919.
16. Walker: Clin. Soc. Trans., London **38**:80.

The causative organism in cases of acute meningo-myelitis is suggested by a positive blood culture during the illness, or the determination of inflammatory foci. Evidences of inflammatory reaction in the spinal fluid and positive cultures from fluid taken during the course of the disease are decisive. Postmortem evidence is best obtained by special bacterial stains or by culture from the substance of a pathologic cord. It is to be remembered, however, that the micro-organisms are short lived in the spinal cord. Of debatable importance are those instances in which bacteria in small numbers and of less well recognized pathogenicity are cultivated, either immediately antemortem (twenty-four hours) or postmortem, from the spinal fluid and blood. Such was the difficulty encountered by F. Buzzard [17] in a case of acute ascending myelitis.

It is well to keep these considerations in mind, especially in cases of toxic or degenerative ascending myelitis, the destroyed tissues of the cord acting as a locus minoris resistentiae for an agonal invasion of organisms by the hematogenic route.

It is not necessary here to enter into the much discussed field of Landry's paralysis and acute polyneuritis. It is enough to state that we will probably never know the nature of the disease which called forth Landry's original description of classes of cases which clinically showed a rapidly spreading motor paralysis, affecting first the legs and, ascending, affecting the muscles of the trunk and arms, and producing death by paralysis of respiration. There were no atrophies, sphincteric disturbances or objective sensory findings, fever was slight, and he was unable to detail pathologic changes by the methods at his disposal. It is probably enough to state that such a class of patients still exists which, with modern necropsy technic, shows slight changes in the brain and cord (F. Buzzard,[17] Gardinier,[18] Hun,[19] Stafford.[20])

The etiology of such cases has been much discussed without any uniformity of opinion. The syndrome has been observed in both toxic states (Barker)[21] and infective states (Buzzard).[22] Associated with a toxic state are the class of cases referred to as acute degenerative myelitis. In this condition more evidences of damage are observed in the cord, especially of a degenerative nature, with complete absence of signs of inflammation (Globus).[23]

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17. Buzzard: Brain **30**:65, 1907.
18. Gardinier: J. Nerv. & Ment. Dis. **41**:640, 1914.
19. Hun: New York M. J. **53**:609, 1891.
20. Stafford: Lancet **7**:1172, 1915.
21. Barker: Contributions to the Science of Medicine, Baltimore, The Johns Hopkins Press, 1900, p. 913.
22. Buzzard: Brain **26**:94, 1903; ibid. **30**:77, 1907.
23. Globus: Ascending Toxic Myelitis, J. A. M. A. **69**:816 (Sept. 8) 1917.

Patients with a progressive polyneuritis may present symptoms of an ascending type of paralysis but clinically differentiated from the complex of Landry by pain along the course of the nerve trunks, sensory and sphincteric disturbances. These cases present marked inflammatory and degenerative changes of the peripheral nerves and are correctly classified as acute toxic or infectious polyneuritis (Hunt).[13]

Wickman [24] showed that the fatal cases of poliomyelitis ended with the same clinical picture as Landry's paralysis and that many cases reported with myelitic changes were acute poliomyelitis.

With these remarks on the allied condition of acute ascending myelitis, we now return to the point at issue in Case 2. The prodromal symptoms, fever and leukocytosis, suggest an acute infection. They are not dissimilar to those in Case 1. The negative blood culture is of importance but not conclusive in ruling out a septicemia. There were no signs of a suppurating process, internally or externally, which might have been classed as a primary focus.

The evidence of an inflammatory reaction in the spinal fluid suggested a meningeal involvement, but the causative organism was not demonstrable in smears from the fluid sediment and the cultures remained sterile. Furthermore, no organisms were seen in specially stained preparations from the cord sections. If, as Buzzard has suggested, acute ascending meningo-myelitis is a more correct pathologic term, and surely there is evidence enough in this case of the meningeal element, why then do we fail to find the causative micro-organism? Is it because, as Homen has shown, the organisms tend to die out quickly in the pathologic cord, or as Strumpell,[25] Marinesco and many others have suggested, the process is hemotoxic or lymphotoxic in origin? Lastly, are we here dealing with an ultramicroscopic organism?

As was previously stated, the fact that the virus of poliomyelitis can under exceptional conditions cause more extensive lesions in the cord than are commonly described is not a novel idea. Taylor [26] years ago in discussing a case reported by Putnam,[27] which showed inflammation and degeneration of the gray and white matter of the cord, questioned the diagnosis. He concluded that the case could with difficulty be put in the category of acute poliomyelitis.

Batten and Holmes [28] in material from three cases of poliomyelitis showed that there was degeneration of the proprio-spinal fibers in the ventrolateral columns of the cord. This was not an attempt to prove

24. Wickman: Nervous and Mental Diseases, Monograph Series **16**:61, 1913.
25. Strumpell: Neurol. Centralbl. **20**:415, 1901.
26. Taylor: J. Nerv. & Ment. Dis. **29**:448, 1902.
27. Putnam: J. Nerv. & Ment. Dis. **10**:14, 1883.
28. Batten and Holmes: Brain **35**:259, 1912.

that the virus of poliomyelitis attacks the white matter of the cord, but an endeavor to trace the endogenous fibers of the cord by the knowledge that poliomyelitis produces its destructive effects chiefly on the gray matter, with resulting secondary degeneration. In the severely affected regions, there were degenerated fibers scattered diffusely throughout the ventrolateral columns, and thus no definite systematization could be recognized.

Harbitz and Scheel [29] report a case of poliomyelitis showing a diffuse inflammation of the cord and medulla with a more acute, swollen and softened hemorrhagic focus localized in the region of the tenth and eleventh dorsal segments and, microscopically, intense inflammatory reactions in both the gray and white substance. They believe their case represents a transitional form between acute myelitis and poliomyelitis, and that certain cases of acute myelitis should pathogenetically and etiologically be placed in a class with acute poliomyelitis.

Skoog [30] reports a case of poliomyelitis of the transverse myelitis type. The clinical condition suggested a transverse lesion in the dorsal region and the patient died four months after the onset of the paralysis with increasing bed sores, cystitis and other complications. Antemortem cerebrospinal fluid showed a mild pleocytosis with 90 per cent. lymphocytes, very marked fibrin formation, and no organisms. The blood culture was negative. The cord presented marked thickening of the meninges and the lower five dorsal and lumbar segments showed cylindrical microscopic cavities extending up and down in both the white and gray matter, especially marked in the posterior columns. The softening in the sacral and coccygeal region, from which staphylococci were grown, probably did not represent a factor in the primary condition.

There are a few instances reported as poliomyelitis of unusual type and the extent of the process in the white substance of the cord is evident from their symptomatology. Anderson and Frost's [31] case gave symptoms of upper motor neuron damage, i.e., spasticity of the paralyzed limbs, exaggeration of reflexes and no atrophy. The patient's serum protected a monkey from a fatal dose of a known virus according to the method described by Flexner and Lewis.[32]

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29. Harbitz and Scheel: Epidemic Acute Poliomyelitis in Norway in the Years 1903 to 1906; Results of Anatomic Investigation of Nineteen Cases of Acute Poliomyelitis and Kindred Conditions, J. A. M. A. **49**:1420 (Oct. 26) 1907.

30. Skoog: Acute Poliomyelitis, Transverse Myelitis Type, J. A. M. A. **59**: 764 (Sept. 7) 1912.

31. Anderson and Frost: Abortive Cases of Poliomyelitis: An Experimental Demonstration of Specific Immune Bodies in Their Blood Serum, J. A. M. A. **56**:663 (March 4) 1911.

32. Flexner and Lewis: Experimental Poliomyelitis in Monkeys: Seventh Note: Active Immunization and Passive Serum Protection, J. A. M. A. **54**:1780 (May 28) 1910.

Sach's [33] case presented symptoms of a transverse myelitis in the high dorsal region with retention of urine, almost complete anesthesia from the tip of the xiphoid distally, with paralysis of the lower extremities, increased reflexes, exhaustive bilateral ankle clonus and a positive Babinski's sign. The spinal fluid showed a pleocytosis and the cultures were sterile. Blood and spinal fluid Wassermann tests were negative. Ten days before discharge, her blood serum rendered the virus of poliomyelitis inactive and protected a monkey, thus giving a neutralization test which in most instances is specific. Such a demonstration justified the inference that the patient had passed through the infection of acute poliomyelitis.

At a later date Sachs [34] in a paper on the infectious forms of acute transverse myelitis reports two other cases which were considered by him as poliomyelitis. Both patients recovered. In the same paper he raised the question as to whether we are to suppose that cases of acute ascending paralysis of infectious origin are in any way identical with acute poliomyelitis.

By analogy to a small number of reported cases, by reference to some of the experimental and pathologic data, and by the clinical methods at our disposal, an attempt has been made to show that not all cases of acute infectious myelitis come under the category of a bacterial or bacterial-toxic origin.

There is possibly an isolated group of these cases, clinically presenting symptoms of transverse myelitis and etiologically dependent on the virus of poliomyelitis.

The same possibility is suggested regarding cases of acute ascending myelitis similar to Case 2, here reported. With symptoms of an acute infection, signs of a regular step-like ascent of the process in the cord and bulb, and spinal fluid the character of which is possible with poliomyelitis and necropsy findings which did not aid in establishing a bacterial etiology, we are justified in Case 2 in further considering the cause. The regularity of the ascent is a point against ascending myelitis of bacterial origin as was so correctly demonstrated by Gower's [35] case. The blurring of the left optic disk in our case, a rare phenomenon in poliomyelitis, is not an infallible point of differentiation.[36] The fact

33. Sachs: J. Nerv. & Ment. Dis. **39**:747, 1912.

34. Sachs: Trans. Assn. Am. Phys. **29**:81, 1914.

35. Gower: Clin. Lect. Dis. Nerv. Syst., 1895.

36. During July, August and September, 1920, in the medical wards of the Peter Bent Brigham Hospital, five cases of poliomyelitis proved by necropsy findings were observed. In one both optic disks showed diffuse blurring of the margins and haziness of the nerve heads. This rare phenomenon of poliomyelitis is important as regards Case 2, under discussion.

that this case immediately preceded a considerable epidemic of poliomyelitis in Boston lends weight to the idea that it was due to a similar infection.

The clinical diagnosis, acute ascending myelitis, means little in these cases, principally because we do not know the cause. Little advancement will be made either in classification or treatment without learning more about etiology.

We regret to state that our evidence is incomplete. Had we pathologic evidence of poliomyelitis after subsequent injection of the cord emulsion into a monkey, our case would be complete. Unfortunately this procedure was not attempted.

As Flexner has remarked, "In my opinion there is no test that is conclusive, pathologically speaking, except the histology of sections prepared from the central nervous system." The pathologic demonstrations complete, therefore, the discussion regarding this case.

CONCLUSIONS FROM PATHOLOGIC EXAMINATION

Case 2 presents many interesting points for study. The pathologic picture agrees with the clinical in that there can be demonstrated an acute ascending affection of the spinal cord with less marked lesions in the basal ganglions and cerebral cortex. Acute myelin degeneration in the cord and spinal radices is the most conspicuous lesion, hemorrhages into the gray and white matter and perivascular lymphocytic reaction are next in prominence, and least marked are degenerative changes in the gray matter and nuclei of the pons. The meningitis present is such as is usually found in acute anterior poliomyelitis and other infectious states involving the central nervous system. The gross and microscopic findings in cord and brain are those of an infectious process, with the virus showing a great predilection for nerve tissue. The vascular lesions are not pathognomonic of any one condition.

Preceding by about a month an outbreak of acute anterior poliomyelitis in Boston and the vicinity, this case necessitates careful scrutiny to remove any doubt as to whether or not it has any relation to the cases of poliomyelitis which followed it. The prominence of the cord lesions would be sufficient to place it in the Heine-Medin group. Degeneration of white matter in poliomyelitis has in practically all epidemics been conspicuous by its absence or insignificance. With a view of determining whether this lesion was duplicated in the epidemic of July and August, the cords of several cases were studied by Marchi's method. These were fulminating cases, the entire illness lasting from three to six days. In one of them as much damage was found in the white matter as was found in the case under discussion.

The diagnosis of choice is acute diffuse ascending myelitis. While it is impossible to be sure that in this case we are dealing with the result of an infection by the poliomyelitis virus, it is equally difficult to deny possibility in view of the clinical picture, pathologic findings (although quite different in order of prominence of the lesions from poliomyelitis), and its occurrence so little in advance of a sizable epidemic of acute anterior poliomyelitis. Also the idea of "change in type of disease," recently emphasized by Sir Humphrey Rolleston, surely must be as applicable philosophy in this disease as in others.

OSSIFICATION OF THE MENINGES*

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"Death seems to be the only object of hope and consolation in all such deplorable cases, as the unhappy patient must be frequently subject to pains, spasms, and occasional dereliction of the faculties, both of body and mind, which . . . are likely to terminate . . . in palsy, mania, idiotism or other maladies more grievous than death itself." Such was the comment of Dr. J. Smith [1] in a letter, written in 1780, to William Hunter, referring to ossification of the meninges, which he had discovered, postmortem, in a recent case. In that period of medical practice, such a pathologic finding sufficed to explain the most grotesque and most divergent clinical findings. Paralyses, chorea, epilepsy, a wide range of psychotic manifestations and feeble-mindedness, constitute a few of the maladies to which Dr. Smith referred. Many case reports were published in an attempt to interpret countless symptoms, the true nature of which the medical man of that period could not otherwise explain, because of what we now know to have been insufficient knowledge of anatomic and physiologic relationship.

Again, the accidental discovery of bony tumors, plaques and extensive ossification of the meninges in persons who had certainly died of systemic disease, and who had not previously manifested cerebral symptoms, led to widespread discussion of the subject.

The literature shows an active interest on the part of medical men, especially during the first half of the nineteenth century; but it is most probable that the celebrated lectures of Virchow, insisting on the benign character of these pathologic changes, checked the impetus of pseudoscientific interpretation, and, as a result, interest lagged, and reported cases since 1870 have been comparatively few. Whether the percentage of occurrence has been actually less, or the presence of meningeal ossification has been observed and noted as unimportant is a matter of question. The fact is that it is most difficult to find any mention of the subject in our leading textbooks on neurology and

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* Read at the forty-seventh annual meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

1. Smith, J.: The History of an Extraordinary Affection of the Brain, Med. Obs. Soc. Phys., London **6**:180-185, 1784.

pathologic anatomy. Oppenheim mentions it only to state that ossification is common, and may be extensive, even to the point of enveloping the brain, but that it has no clinical significance.

While none of us today has the temerity even to suggest that bony transformation of the meninges may produce the cerebral mischief mentioned, we are impressed with the possible significance of this finding in certain cases. A careful study of the literature has revealed most remarkable and almost unbelievable osseous changes. While, for the most part, the pathologic reports may be accepted as trustworthy, it is evident that the conclusions in many instances were incorrect. Frequently such findings as milky exudates, hemorrhagic areas, "preternatural congestions," areas of softening, free blood and even pus in the ventricles, indicate at once the probability that the ossified areas were only coincidental changes.

But, in the broader aspect, it is almost certain that the location, extent, depth and other characteristics of osteophytic processes described in the early literature would present to us, in isolated instances, definite signs of intracranial disturbance. Today we are quite aware of the manifest symptoms that may arise from a small irritative lesion of the cerebral cortex or the overlying meninges, especially in certain areas. On the other hand, we are aware that extensive infiltration of the brain substance may occur without giving rise to significant symptoms. Hence it may be of interest to summarize briefly the pathologic changes that have been described to date, in the earnest hope that, in the light of our more advanced methods of examination and interpretation, their occasional clinical importance may be noted.

The falx cerebri easily assumes first place as the most common site. The regions of the dura alongside the superior longitudinal sinus, and the tentorium are next in frequency of occurrence. All areas of the convexity, however, may be affected.

The dura is frequently involved alone, or may be the seat of a process which involves the other tunics as well. Again, ossification may have its origin in the pia-arachnoid, leaving the dura free.

The meninges may be the seat of isolated plaques, minute and multiple, or of clusters, irregularly placed or united to form in plates extending anteroposteriorly the length of the brain and laterally over the convexity so that the brain is practically encased in a bony sheath.

The osseous formations may be pyramidal in shape and multiple, and the convolutions are seen to be molded exactly to the opposing surfaces. Solid masses of bone, the size of a hazelnut and larger, may develop from the inner surface of the pia-arachnoid and become embedded in the cortical substance between the convolutions. These have been invariably associated with corresponding changes in the meninges overlying them.

The thickness of the specimens reported varies between a millimeter to a centimeter, perhaps in the same plaque. The outer surfaces are invariably smooth and glistening, while the inner surfaces are always rough and jagged. Stalactite processes are common, and may be so pointed that the outer tunics are pierced and the cortex invaded for a considerable distance. This is a peculiarly significant feature from the standpoint of symptomatology.

The meninges involved usually manifest a congestion of the vessels, and, especially in those cases in which there has been a history of trauma, adhesions are frequently observed.

A theoretical discussion of the relationship of calcification to ossification will not be entered into; nevertheless it is well to note that many of the published cases have demonstrated true bony changes. We know that areas of the meninges or brain substance that have become calcified through the medium of extravasated blood due to trauma or from any other cause may become ossified. We know that in the presence of osteoblasts such changes may take place in other parts of the body, such as the heart, kidney, pleura and eyeball. Whether such metaplasia in the brain and its coverings is made more readily possible because of the embryologic functions of the dura, or whether through a specific action on connective tissue cells calcium leads to ossification, are questions apart from the purpose of this communication.

Despite the absence of postmortem confirmation, the following case, representing extensive ossification of the meninges, with certain evidences of focal irritation or pressure, is worthy of report. To our knowledge, it embodies the first attempt to study, according to our present day methods, the clinical manifestations of such a pathologic process.

REPORT OF A CASE

History.—Mrs. X., aged 49, married, born in the United States, a housewife, came under observation in March, 1921, through the courtesy of Dr. Evan Evans. She complained of attacks of vomiting, severe headaches, insomnia, languor, and lack of endurance. These symptoms had been particularly prominent for nine or ten years. The patient believed that with the exception of the vomiting, her condition had become gradually worse.

Her mother died at 70, having had trigeminal neuralgia, pernicious anemia and some cardiac disturbance. Her father died of "rheumatism" combined with anxiety. Two brothers were well, and her husband and two children were quite well. There was no history of a similar affection in the collateral branches. As a child the patient was never very robust. She was always considered anemic, and between the ages of 12 and 14 had had severe "fainting spells" but no convulsions. She had had all of the usual childhood diseases, including measles in a particularly serious form. Tonsillotomy was performed during adolescence. Menses appeared at the age of 13 and had always been regular, rather profuse and painful. An attempted curettage and cervical

Fig. 1.—Roentgenogram of case reported. Mottling of inner surface of skull which stereoscope indicates is not connected with the base and which is probably due to ossification in the meninges.

repair failed twenty-five years ago because the pulse became so weak under the anesthetic that the surgeon could not continue the operation. There was a vague possibility that a miscarriage occurred twenty-three years ago.

Present Illness.—Since her pregnancies the patient had not been as well as formerly, but during the past ten years especially she had suffered severely with the symptoms enumerated. The care of her mother who died eight years ago had involved a great deal of anxiety and responsibility.

The vomiting spells first occurred about nine years ago at a time when the patient was acutely anxious concerning her son's health. Since that time they had been fairly constantly present, sometimes occurring every day for perhaps two weeks, with free intervals of several weeks occasionally. Three months had been the longest interval during which she had not vomited. Nausea usually preceded the attacks. They occurred during sleep as well as at any time of day. They bore no relation to the taking of food. They were occasionally so severe and prolonged that the patient fell asleep from sheer exhaustion. Walking and exercise in general had repeatedly increased the frequency of her attacks. Excitement had never precipitated them.

The headaches described as neuralgic in type, usually but not always unilateral, had persisted for years. There was no periodicity in their appearance, but they were always present at the menstrual periods as well as at other times. The usual location was frontal, with dull aching and drawing pain sometimes referred to the face and throat. Excitement had frequently precipitated and intensified them. The patient also described "sick headaches" associated with vomiting which she characterized as quite different from the neuralgic type.

Insomnia had existed during the same period of years. Five hours was the average amount of sleep obtained. The lack of endurance and languor had been most troublesome and had led to very much circumscribed activities. They have never yielded to the effects of tonic stimulation.

The general physiologic status was good. The bowels were regular. There was frequency of micturition but never incontinence. Digestion, excluding the vomiting attacks, was normal. There had been no especial change in weight during the past ten years.

Examination.—Abnormal Attitudes and Deformities: There were no abnormal attitudes. There were no deformities, except a webbing of the second and third toes of each foot.

Gait: This was normal in all its phases. Associated movements were normal.

Coordination: Equilibratory: Standing with feet together was done well with eyes open and closed. The patient stood on one foot poorly, right and left.

Nonequilibratory: Finger to nose tests were done well, right and left, with eyes open and closed. Finger to finger, and each finger to thumb tests were normally performed. Heel to knee tests were normal. There was no real adiodokokinesis, but the movements of the left upper extremity were awkward compared with those of the right. There was no dysmetria.

Skilled Acts: There was no apraxia. Handwriting was normal. Speech was entirely normal.

Abnormal Involuntary Movements: There was slight digital tremor on each side, greater on the left than on the right. There were no twitchings, choreiform movements or other subnormalities.

REFLEXES *

Deep †	Right	Left
Pectoral	2+	2
Biceps	3+	2
Triceps	3+	2
Radial	3+	2
Ulnar	2	2
Wrist	2	2
Suprapatellar	2	2
Patellar	2	2
Achilles	2	2
Ankle clonus	0	0
Superficial		
Ciliospinal	1	1
Supra-umbilical	1	1
Supra-pubic	1	1
Upper lateral abdominal	1	1
Lower lateral abdominal	1	1
Plantar flexion	1	1
Babinski	0	0
Fanning	0	0
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schäfer	0	0

*0—absent. 1—present. 2—active. 3—very active.

†The deep reflexes have been repeatedly examined on several occasions, and the same inequality has been definitely and invariably found.

Muscle Strength: In all muscle groups muscle strength was much below the average normal. There was, however, no disparity between the two sides that could be determined. The weakness was noticed particularly in the fingers, wrist and forearm muscles, especially in opposed flexion and extension.

Muscle Status: Volume and contour were normal. There was no atrophy or hypertrophy. The consistency was soft and flabby. The tone was normal. There was no perceptible myotatic irritability or myoidema. Electrical tests were not made.

Abnormal Associated Movements: There was a definite Hoffmann sign on the left constantly. It was obtained on the right at times

General Sensory Examination: Touch: Acuity, localization and discrimination were normal throughout. Pain: Superficial and deep pain were normal. Temperature: Moderate degrees of heat and cold were accurately determined. Vibration: Vibratory sensation was normal in the upper and lower extremities. Muscle-tendon: The sensibility was normal in the toes, fingers, forearms and arms.

Stereognosis: The size and form of objects were correctly noted.

Cranial Nerves: Optic, Oculomoter, Trochlear and Abducens Nerves and Optic Apparatus: Visual acuity was not tested. The fields were normal to

rough tests. The fundi were normal; the pupils were equal, regular, central and moderately dilated. They responded briskly to light and convergence. There was no strabismus. no ptosis, no nystagmus, no exophthalmus or enophthalmus.

Acoustic and Ear: Acuity of hearing was normal for a watch tick. Air conduction was better than bone conduction on both sides. The Rinne test was normal.

Fig. 2.—Case not accessible to clinical study showing similar osseous changes. Areas of meningeal density which are probably due to ossification.

Trigeminus and Mouth: The motor fifth nerve was normal. The corneal reflex was brisk on each side. There were no abnormalities of the mouth or of the teeth worthy of note.

Facial Nerve and Face: In conversation there was a marked weakness of the lower two thirds of the left side of the face. In smiling this weakness was still apparent. In forced movements, however, such as showing the teeth, and in hearty laughter the weakness was no longer manifested.

Articulation of the labials was normal. Associated movements of the face, eyes and head were normal.

Glossopharyngeus, Vagus Nerves, Pharynx and Larynx: Taste was not examined, but was probably normal. The uvula was normal in position and rose in midline to phonation. Palatal and pharyngeal reflexes were normal. Phonation and swallowing were normal.

Spinal Accessory Nerve: No abnormality in the function of the trapezii or sterno-cleido-mastoids could be elicited.

Hypoglossal Nerve and Tongue: The tongue protruded in the midline; it moved well in all directions and presented no trophic disturbances.

Mental Status: The patient was of an extremely intelligent type. She was alert, of a genial disposition and decidedly not introspective. Her mental life was one filled with detailed care and management of her family and home

Fig. 3.—Distribution of these ossifications through the frontal areas.

affairs. She was not irritable, in the ordinary sense, although the usual disturbances of a large household were frequently a source of annoyance which she did not express externally. There was no emotional instability. In fact, the patient's husband considered her especially well balanced. Attention and memory were not defective. There were no phobias, compulsions, hallucinations or delusions.

Cranial Morphology: The skull was normal to palpation. There had been no noticeable increase in size.

Systemic Examination: Tegumentary: Her hair was normal in color, amount, texture and distribution. There was no pigmentation of the skin or vasomotor disturbances of any kind. Adipose tissue was rather excessive, but evenly distributed.

Glandular: The thyroid gland was normal. The tonsils were small and almost invisible. There was no palpable adenopathy.

Cardiovascular: The heart was normal. Systolic pressure was 130. The pulse rate was variable, averaging 80.

Respiratory: The lungs were normal.

Gastro-Intestinal: The liver and spleen were not palpable. Palpation and percussion was otherwise normal. (See roentgen-ray report.)

Genito-Urinary: The patient had a rather excessive leukorrhea, but no pelvic signs or symptoms. As stated in the foregoing, micturition was sometimes urgent.

Fig. 4.—Section of osteophyte of the falx cerebri. Specimen submitted by the courtesy of Dr. J. H. Larkin, pathologic department, City Hospital.

Laboratory Examination.—Urine Analysis: The findings were: appearance, very turbid; color, pale amber; reaction, acid; specific gravity, 1.028; albumin, trace; sugar, absent; acetone, absent; diacetic acid, absent; indican, normal; urea, 2.4 per cent.; bile pigments, absent; blood pigments, absent.

Microscopic Examination: The examination revealed: casts, none; cylindroids, none; mucus, a few threads; amorphous, few urates; crystals, none; pus, a few cells; red blood cells, none; bacteria, numerous motile bacilli (contamination); epithelia, few squamous cells.

Blood Wassermann Reaction: The extracts used were crude alcoholic and cholesterinized. The result was negative.

Blood Chemistry: The following determinations were made in 100 c.c. of blood: uric acid, 3.2 mg.; urea nitrogen, 18 mg.; urea, 39.5 mg.; creatinin, 2.6 mg.; sugar, 125 mg. or 0.125 per cent. The examination showed slight nitrogenous retention and hyperglycemia.

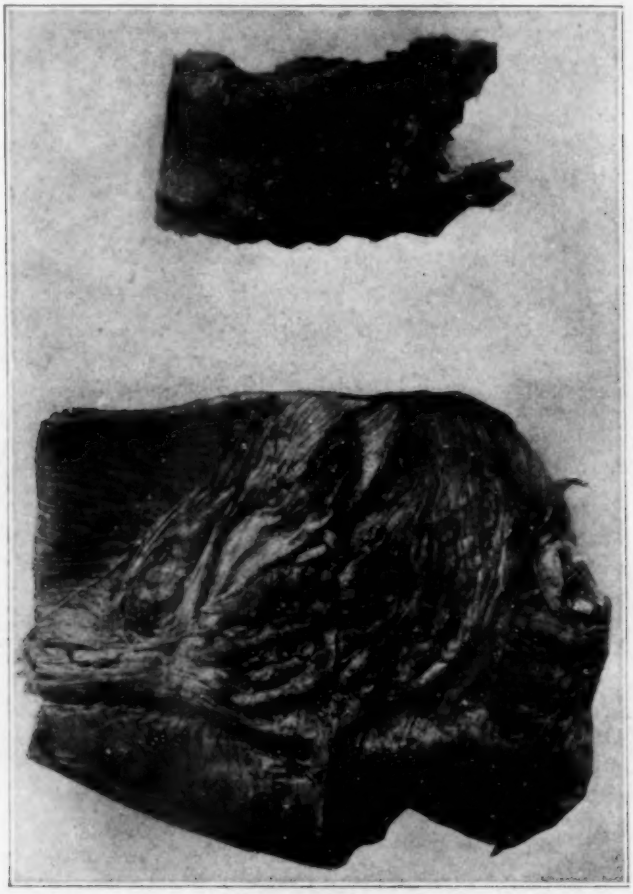

Fig. 5.—Osteophytes of the dura, near superior longitudinal sinus. Note jagged contour of internal surfaces. Microscopic examination revealed true bone. Specimen submitted by courtesy of Dr. Douglas Symmers, Director of Laboratories, Bellevue Hospital.

Complete Blood Count: The blood count revealed: erythrocytes, 4,200,000 per cubic millimeter; leukocytes, 6,000 per cubic millimeter; hemoglobin, 76 per cent.; color index, 0.9 per cent. Differential Leukocyte Count: polymorphonuclear neutrophils, 59 per cent.; small lymphocytes, 38 per cent.; large lymphocytes, 0; large mononuclear leukocytes, 0; transitional leukocytes, 1 per cent.; eosinophils, 2 per cent.; basophils, 0.

The examination revealed a moderate diminution in the percentage of hemoglobin and in the number of red corpuscles, with a fair number of leukocytes and practically a normal differential count.

The red corpuscles were quite uniform in size and shape.

Gastro-Intestinal Serial Roentgen-Ray Taken 1913: Roentgenograms showed residue after six hours. The pylorus was apparently filled out properly. The cecum was rather low, the stomach high. .

Skull: Roentgen-ray examination of the skull showed it to be of normal size with a slight increase in its thickness and density. The outer surface seemed to be quite normal, but the inner surface of the frontal and parietal regions was definitely roughened. Stereoscopic plates as well as the anterior ones showed that this was not confined to the longitudinal sinus but was distributed over the entire frontal bone and over an irregular area in the parietal. The stereoscope would further indicate that these processes were of about the same density as the bone and that they projected into the cavity. The sella was not disturbed, and there were no indications of increased intracranial pressure.

Summary of Essential Findings.—These were: history of headaches over a period of nine years; vomiting, occurring at any time of night or day at irregular intervals, aggravated by exercise; pathologically increased deep reflexes of the right upper extremity and bilateral Hoffmann's sign; facial weakness of the central type on the left; roentgenogram of the skull revealed extensive ossification of the meninges.

In a careful and prolonged study of the patient we have been convinced that the signs and symptoms described in the foregoing are due directly to the pathologic condition of the meninges. All other abnormal changes in the skull that might simulate meningeal ossification, including syphilis, carcinoma, tuberculosis and Paget's disease have been easily eliminated.

While our belief that meningeal ossification can be diagnosed during life has not yet been substantiated by postmortem confirmation in this case, it is our conviction that careful investigation in this direction will prove ultimately the occasional clinical importance of this phenomenon.

DISCUSSION

Dr. Beverley R. Tucker of Richmond said that he thought this presentation was of great interest, and he briefly reported a case of ossification of the spinal meninges.

About four years ago he had seen the patient and made a diagnosis of spinal tumor, although the central level was quite indefinite. Her pain, however, was severe and radiated around her body. She had a spastic paraplegia which seemed to be progressive. Her symptoms dated back for about a year. A radiograph of the whole spinal region failed to show anything unusual. Dr. Tucker supposed that it was possibly harder to pick up plaques of this kind on the cord meninges than it was on the brain. Dr. Stuart McGuire of Richmond operated on the patient, and, much to their surprise, found ossified plaques—some of them separate and some overlapping. They were a little larger than the little finger nail; however, some were larger and some were

smaller. They exposed an area of about three and a half vertebrae, and although ten of these plaques were removed, they felt that they had not removed all of them. The case was closed up and the patient had been under his observation ever since. He thought the interesting part of it was that the spastic paraplegia had progressed and seemed to have extended up the cord. The spinal cord in the region of most of the plaques had dwindled to about half of its size, showing atrophy; it took on a conelike shape. The specimens of these plaques were sent to the laboratory and were reported as cartilage or bone.

It had been about four years since the operation and one arm was considerably involved by pain and some disturbance of motion, and the other arm was slightly involved. This woman had never shown any cerebral symptoms, as far as they could elicit. He had hesitated to advise operation but had a surgeon examine her with a view to the possibility of another operation to remove more of these plaques, but the prognosis seemed so uncertain that nothing had been done.

Dr. Ernest Sachs of St. Louis said that these ossifications were particularly interesting to him because of the great difficulty of differentiating endothelioma, which are known not infrequently to come from the dura and which not infrequently present a picture similar to those which are seen with ossification, and he would be very much interested to know whether Dr. McKendree could throw some light on how to differentiate these ossifications from ossified endothelioma.

He had operated on two patients, in both of whom he and Dr. Moore, the otologist, believed that there was more of a tumor than the calcification in the dura.

Dr. Charles A. Elsberg of New York said that the Association ought to be much indebted to Dr. McKendree for having brought forward this interesting subject. He had been interested and often much worried because he had seen so many ossifications and calcifications in the meninges and had not understood their significance. He had asked himself whether they could give rise to symptoms.

In many instances of degenerative disease of the central nervous system, and especially in atrophic spinal cords, these calcified plaques often exist in large numbers. There may or may not be a history or the evidence of trauma. The question was whether these calcifications were not entirely secondary to the disease of the nervous system and whether they could give rise to symptoms or influence the changes in the diseased nerve tissue.

Dr. Charles A. McKendree of New York, in closing, said that in reading the literature of the subject, he had found ossified and calcified plaques of the spinal meninges mentioned frequently. Also, in the lower animals, especially the dog, progressive spinal paralysis attributed to ossification of the dura and pia-arachnoid had been reported by French veterinarian schools.

In discussing the matter with several prominent pathologists he had learned that they were commonly encountering ossified areas in the spinal membranes, but that these lesions were not very extensive and did not have the characteristics of depth and penetration that the cerebral plaques had. Therefore, he would be surprised, in general, to find that they could be as significant in man as the cerebral ossifications.

With reference to Dr. Sachs' question as to endotheliomas, he would say that in the case reported there were none of the usual signs of increased intracranial pressure. The symptoms had endured for at least ten years, and the findings could not justify the diagnosis of cerebral neoplasm.

OBSERVATIONS ON CHARACTER OF THE ONSET OF SPINAL PARALYSIS WITH REFERENCE TO THE SIGNIFICANCE OF THE APOPLECTIFORM TYPE OF ONSET IN CONTRAST TO THE SLOW PROGRESSIVE DEVELOPMENT OF PARALYSIS *

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PHILADELPHIA

I desire to present a few observations on the significance of the variations in the character of the onset of certain forms of spinal paralysis, for I believe that in some instances the nature of the pathologic conditions present may be determined by the mode of onset alone.

Extramedullary spinal tumors are generally found to be either fibromas or endotheliomas. They grow slowly, producing gradually a weakess of the limbs that increases progressively. Spinal thrombosis, on the other hand, is marked by a sudden or apoplectiform onset; it is not a rare condition, and is usually caused by syphilitic arteritis of one or more of the blood vessels of the spinal cord. The sudden onset of spinal paralysis, however, has also been regarded as indicative of hemorrhage within the cord; but spontaneous hemorrhage, or hematomyelia without trauma, is so exceedingly rare as to render its occurrence most doubtful, for it is not probable that normal blood vessels would burst unless they were injured.

Of the cases of spinal tumor recorded here, four were extramedullary fibromas. Early in their growth they gave rise to pain, due to irritation of the sensory nerve roots. Pain may be the first symptom of a pathologic condition and may be present long before the tumor has attained sufficient size to exert pressure on the cord and thus induce motor weakness. In one case (Case 4) pain was complained of five years before motor symptoms were observed; in another (Case 3), pain was present three months before other symptoms developed, whereas in still another (Case 1), the motor and sensory symptoms appeared almost simultaneously.

The prolonged duration of severe unilateral pain as the sole symptom of disease is most difficult of interpretation. It is important to remember that it may be caused by a spinal tumor. This has fre-

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quently been referred to in the literature on the subject, Auerbach [1] having published the description of a case in which this symptom was the only one present.

Some observers assert that the absence of pain is confirmatory evidence that the tumor is an intramedullary, and not an extramedullary, growth. However, in my case (Case 5) of intramedullary tumor—an ependymoma—pain was present, but not until long after motor weakness had developed. Such a distinction, made on this basis alone, is not justifiable, for many instances are recorded of extramedullary tumors that attained considerable size without involving the sensory roots sufficiently to cause pain; this is particularly true if the tumors grow from the anterior surface of the cord. Pearce Bailey [2] has referred to painless tumors of the spinal cord. The statistics of Steinke,[3] however, based on an analysis of 330 cases, show that spinal tumors grow most frequently from the posterior and posterolateral surfaces of the cord.

Elsberg [4] has reported cases of tumor growing from the anterior surface of the cord in which pain was absent. It would seem, therefore, that, at least in the early stages, the presence or absence of pain is dependent on the situation of the tumor.

Marie and Foix [5] have endeavored to show that the distribution of muscular atrophy induced by an intramedullary spinal growth may not be identical with that caused by an extramedullary spinal tumor. In my Case 5 muscular atrophy appeared to be extensive. Despite the importance of these observations, the differential diagnosis of these cases will, in the vast majority of instances, always be extremely uncertain.

Patients who are paralyzed in the lower limbs as the result of tumor of the cord not infrequently complain of severe pain in the legs, in spite of the fact that cutaneous sensation is diminished or even abolished. For example, in Cases 1 and 3 severe pain in the paraplegic limbs was complained of constantly, although there was anesthesia of these parts. Anesthesia dolorosa, on the other hand, caused by irritation of the sensory spinal nerve roots, is a familiar symptom, but is different in character from the painful sensations to which I have referred. Elsberg has mentioned pain in the lower limbs occurring in spinal cord tumor, resembling, in some respects,

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1. Auerbach: J. Psychiat. & Neurol., 1910.
2. Bailey, Pearce: Painless Tumors of the Spinal Cord, J. A. M. A. **63**:6 (July 4) 1914.
3. Steinke, C. R.: J. Nerv. & Ment. Dis. **47**:418 (June) 1918.
4. Elsberg: Arch. Neurol. u. Psychiat., January, 1921, p. 64.
5. Marie and Foix: Rev. neurol., 1914, No. 4, p. 327 (Society Report).

the painful sensations that Förster [6] records in a recent article, and which Nonne, in a discussion of the subject, called a neighborhood symptom, or "fern Symptome." Sargent [7] also speaks of "pain in the legs" caused by tumor of the cervical portion of the cord.

Spontaneous aching pain occurring in regions that are not supplied by the sensory nerves directly affected by the tumor is difficult to explain. It has frequently been suggested, however, that symptoms may arise from certain limitations in the circulation of the spinal fluid caused by occlusions produced by the tumor, together with alterations in the blood and lymph stream within the cord due to pressure; or they may be brought about by disturbances in the vascular supply of the paralyzed limbs themselves, due to loss of vasomotor tone in the peripheral circulation. Holmes [8] has described pain—or central pain, as he calls it—occurring after gunshot wounds, which he attributes in part to swelling and distention of the injured cord due to edema. It is probable that one or all of these factors may explain, at least in part, the occurrence of pain in the lower limbs, even when the tumor is situated on the cervical or on the thoracic portion of the cord.

In most cases of tumor of the cord motor paralysis takes place so gradually that the actual date of its appearance cannot be determined. It generally begins insidiously, with subjective sensations that are, at first, rarely recognized as important, but that are, in reality, the earliest expression of the rapid exhaustion of weakened muscles.

It is the very gradual transition from a normal to an abnormal state that the patient is quite conscious of, but that the examiner is unable to demonstrate as the beginning weakness that constitutes the earliest phase of severe paralysis.

Cases of sudden onset of motor paralysis in spinal tumor must be exceedingly rare, but Förster refers to one case of this kind in his series, although he gives no details.

Müller [9] recorded a case of tumor growing from the upper cervical portion of the spinal cord that had caused a sudden onset of unilateral paralysis and later remission of these symptoms occurred.

In one case of spinal tumor seen by me, but not recorded here, one lower limb that was already moderately weak suddenly became almost completely paralyzed. This might be compared with the sudden onset of paralysis that occurred in two cases of Pott's disease, reported by me,[10] without deformity of the vertebrae, and caused by myelitis due to compression of the cord, in which there was microscopic evidence of thrombosis of the vessels in the spinal cord.

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6. Förster: Deutsch. Ztschr. f. Nervenheilk. **70**:64, 1921.
7. Sargent, P.: Brit. M. J. **1**:37 (Jan.) 1920.
8. Holmes: Osler's Anniversary Volume, 1921.
9. Müller: Deutsch. Ztschr. f. Nervenheilk. **71**:183, 1921.
10. Cadwalader, W. B.: Am. J. Med. Sc. **112**:546, 1911.

Remission of symptoms during the course of tumors of the cord must also be exceedingly rare, although they are not unknown. Thus, Hedinus and Henschen [11] describe a case, confirmed by the necropsy findings, in which there had been a complete remission of motor symptoms for a short time, and Jumentie [12] reported before the Society of Neurology in Paris a most remarkable case of tumor of the cervical portion of the cord that had caused severe paralysis of all four limbs, and in which spontaneous remission had occurred and continued for three years. The case subsequently ran the usual course, and the diagnosis was eventually confirmed by postmortem examination.

No variations in the character of the development of spinal paraplegia could be more strikingly different than is the onset of paralysis in acute softening of the spinal cord caused by obliteration of the spinal vessels. In one (Case 6), paraplegia developed within a few hours; in another (Case 7), paralysis was complete in one hour.

Spiller [13] has recorded a case in which the first symptoms of paralysis developed within fifteen minutes after the man had been lifting heavy blocks of ice, severe paralysis occurring a few minutes afterward. At necropsy thrombosis, with softening in the first thoracic and eighth cervical segments of the cord, was demonstrated. This was attributed to syphilitic infection. A somewhat similar case is reported by Barth and Léri,[14] in which the patient, six months after treatment for syphilis, suddenly developed headache, vomiting and fever; at the end of the second day he was completely paraplegic, and on the third day the right arm became paralyzed and bulbar symptoms appeared. The patient died seventeen days later. Microscopic examination of the spinal cord showed cellular infiltration of the meninges, marked infiltration of the blood vessels, multiple hemorrhages in the anterior horns and obliteration of the central canal. This was, without doubt, a syphilitic process.

Schultze,[15] in a recent article on acute myelitis, points out that syphilitic thrombosis of the spinal vessels is the most important single factor in causing the sudden onset of paraplegia.

Spiller asserts that spinal thrombosis is probably the cause of most of the apoplectiform palsies that occur during the course of myelitis.

The weight of evidence seems to show quite clearly that the sudden onset of spinal paralysis may mark the advent of more than one disease. Perhaps the best known condition in which this occurs is acute epidemic poliomyelitis although other forms of acute myelitis

11. Hedinus and Henschen: Jahresb. u. d. Leistung auf dem Gebiete Neurol. u. Psychiat. **17**:748, 1913.
12. Jumentie: Rev. neurol. **28**:285, 1921.
13. Spiller: J. Ment. & Nerv. Dis., December, 1908.
14. Barth and Léri: Rev. neurol., 1913-1914, No. 19, p. 393.
15. Schultze: Deutsch. Ztschr. f. Nervenheilk. **68**:1, 1921.

may have a similar onset. In a larger number of so-called cases of acute myelitis, however, the affection is due not to an inflammatory process, but to softening from thrombosis of some of the vessels of the spinal cord, particularly in syphilitic infection, a view that was held years ago by Bastian.[16]

The belief formerly held by Gowers, that long-continued overexertion of the lower limbs, associated with exposure to cold and wet, as occurred in my Case 6, might be responsible for spinal paralysis, has been voiced again by Schultze. However, experience in pathology and the exceptional opportunities afforded by the late war for observing the effects of such conditions have not disclosed any reliable evidence that I know of to show that these factors alone are capable of producing acute spinal paralysis in men who were in other respects normal. On the other hand, it is quite certain that long-continued, fatiguing exertion under unfavorable conditions may be the causative factors determining the moment of onset of spinal paralysis in persons already diseased, as, for example, in those with latent syphilitic infection of the nervous system or other spinal cord disease.

The rapid or sudden development of ataxia may occur when the posterior column of the spinal cord is diseased, without actual motor paralysis, and if it is very severe, because of its sudden development, it might be confused with actual motor paralysis. Such acute ataxia was seen by me[17] in dogs whose anterolateral tracts of the spinal cord had been cut for experimental purposes. In the report of these experiments the condition was referred to as a pseudoparalytic state.

The following notes are taken from the case history of a man who already had tabes and who suddenly became extremely ataxic:

Robert T., a letter-carrier, was under treatment in the outpatient department of the Orthopedic Hospital in 1911 for tabes dorsalis. He had the usual classic symptoms of that disease of moderate intensity, but there was very little or no ataxia. He could walk well without assistance, and despite advice to the contrary he was still pursuing his regular vocation. Not feeling as well as usual and complaining of his legs being tired, he entered a restaurant to get a cup of coffee, sitting on a high stool at the lunch counter. On getting down from the stool his legs suddenly gave way, and he fell to the floor. He stated that he was unable to walk, and had to be assisted. Subsequent examination in the ward of the hospital, only a few days later, disclosed no additional evidence of tabes or other spinal cord disease beyond strikingly severe incoordination of the lower limbs. He was entirely incapable of walking alone. As tested against the resistance of the examiner, the muscular power of the lower limbs was fairly good. After prolonged treatment, chiefly by rest and diet, and reeducational movements, he was able to walk with the aid of two canes, and though the ataxia persisted, there was no motor paralysis.

16. Bastian: Lancet **2**:1531, 1909.

17. Cadwalader, Williams B., and Sweet, J. E.: Experimental Work on the Function of the Anterolateral Column of the Spinal Cord, J. A. M. A. **58**:1490 (May 18) 1912.

In this case extreme ataxia had at first been mistaken for motor weakness. Without a postmortem examination in this case the actual character and extent of the alterations within the spinal cord must necessarily remain undetermined, the clinical signs indicating only posterior column disease.

In another case coming under my notice the patient, who had been regarded as healthy, during the stress of excessive fatigue and exhaustion incident to warfare rapidly developed severe ataxia, the symptoms of tabes, which had not been previously suspected, having remained in abeyance until this time.

The notes of the following case show that ataxia can develop acutely in the lower limbs in a man suffering from posterolateral sclerosis of the spinal cord associated with severe anemia:

Mr. H. was admitted to a private ward at the Presbyterian Hospital in 1920, and gave the following history:

He had been annoyed for some months by a sensation of numbness in his feet and hands and a general feeling of weakness, particularly after the exertion of walking to his office. At that time, Dec. 15, 1919, he had been examined by his physician, who found that his tendon reflexes were absent. The case was erroneously regarded as one of tabes. About February, 1920, he stated that the numbness in his limbs was increasing, and that his legs felt weak—so much so that he remained at home, although he was still able to walk. On February 9, he was unable to walk, even to take a step, and could not stand erect without assistance, due to extreme incoordination of the lower limbs. On examination, the tendon reflexes were found to be absent; the muscular power of the lower limbs as tested against resistance was remarkably good, but he was unable to recognize the position in which the toes, and even the feet and lower portions of the legs, were placed. Vibratory sensation of the legs and arms was absent. The blood count showed: hemoglobin, 39 per cent.; erythrocytes, 2,440,000; leukocytes, 5,200. Gastric analysis showed an absence of free hydrochloric acid.

The alterations of the cord that are associated with these severe types of anemia, in my opinion,[18] are caused by a toxemia, and are not entirely dependent on the anemia itself, that is, using the term anemia in the sense of indicating only a diminution in the quantity of normal blood.

Such rapid development of extreme ataxia is, in my experience, most uncommon, but these examples are recorded here merely to permit a contrast to be made with true spinal paralysis.[19]

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18. Penna. M. J., July, 1921, p. 692.

19. Since the preparation of this paper, an article has been written by Guillain (Bull. de l'Acad de méd., Par. **85**:732 [June 28] 1921), in which he refers to acute ataxia developing in the course of a few hours without any muscular paralysis following an exceptional physical strain in cases of tabes.

Minor (quoted by Oppenheim) states that, of the many cases of hematomyelia observed by him, in only two cases did hemorrhage occur spontaneously. In all the others trauma seemed to be the etiologic factor.

Oppenheim [20] attributes nine-tenths of all cases of hemorrhage to trauma, although he recognizes the fact that in certain affections in which the blood vessels are weakened by disease, for example, in arteriosclerosis, hemophilia and purpura, spontaneous hemorrhage may occur. H. Fabritius [21] reported two cases that came to necropsy in which paralysis associated with fever and other symptoms of acute infection developed suddenly. In the first case he found acute softening of the cervical cord, with thrombosis of the veins and hemorrhage in the anterior median fissure. In the second case hemorrhage took place in the substance of the thoracic portion of the spinal cord, and there were, in addition, perivascular infiltrations, etc. He regarded both as cases of acute myelitis and pointed out that in the second one spontaneous hemorrhage had occurred. These cases seem to show that thrombosis and minute hemorrhages may occur in the myelitis of acute infectious diseases, with rapid development of paralysis. The so-called hemorrhagic type of acute myelitis is generally recognized as the result of an infectious process, but it differs markedly from, and should not be confused with, intraspinal hemorrhage or hematomyelia the result of trauma. Hematomyelia probably never occurs without either trauma or vascular disease, for, as has been stated, it seems most improbable that normal blood vessels could burst unless they were injured or diseased.

REPORT OF CASES

CASE 1.—*Fibroma of the fifth and sixth cervical segments of the spinal cord that had caused gradually increasing weakness of all four extremities. Severe pain in back and in legs. Upper limbs held in the Thorburn position.*

History.—William S. (Lab. No. 563), admitted on March 6, 1911, to the service of Dr. Spiller. The patient's present condition began about November, 1908, with a feeling of heaviness and weakness of the lower extremities. For some time he had had pain in the left upper limb, particularly during the night. These symptoms continued and increased steadily, until five weeks previous to admission, when he was compelled to give up work and remain at home. Recently he had complained of marked pain in the lower limbs on rising in the morning. The weakness of his lower limbs had gradually progressed, and he stated that he was unable to move his legs.

20. Oppenheim: Textbook of Nervous Diseases, trans. by Alexander Bruce, 1911.

21. Fabritius, H.: Zur Kenntnis der akuten Myelitis und der sogenannten spontänen Hämatomyelie, Arb. a. d. pathol. Inst. a. d. Univ. Helsingfors, N. F. **1**:125, 1912.

Physical Examination.—The patient lay with the forearms flexed on the arms so that they were at an acute angle, the hands resting on the chest, just above the breast, in the Thorburn position (Fig. 1). The forearms and hands were wasted to a moderate degree, but the upper arms were not atrophied. Passive extension of the forearm caused pain, probably due to beginning contracture of the biceps tendon. There was complete paralysis of all the muscles of the fingers and hands. Movement was impossible in the fingers and wrists. Extension of the elbow was not possible, owing to complete paralysis of the triceps. Flexion of the forearm at the elbow was possible, but was weak, due to paresis of the biceps, brachialis and brachioradialis. Supination of the forearm was very weak, owing to extreme paresis of the supinator brevis. Adduction of the upper arm was impossible because of paralysis of the pectoral muscles. The deltoid was paretic. The entire inner

Fig. 1 (Case 1).—The credit for recognition of the significance of this peculiar position of the upper limbs is given to Sir William Thorburn, but he mentions that it had been observed and recorded by others previously. He, however, emphasized the importance of this syndrome as indicative of a lesion above the sixth cervical roots. (Sir William Thorburn:" A Contribution to the Surgery of the Spinal Cord," 1889.)

half of the arm on the extensor and flexor sides was anesthetic, as were also the entire hand, all the fingers and the forearm, except a small radial zone beneath the elbow where there was hypesthesia. The middle of the posterior part of the upper arm over the triceps was anesthetic. The region supplied by the circumflex nerve on the upper arm and shoulder was hypesthetic. The wrist reflexes and biceps tendon reflexes of each side were moderately but equally diminished. The triceps reflexes of each side, however, were much exaggerated, though equal. (Here the notes are incomplete, and the report of the subsequent examination stated that the tendon reflexes were abolished

on each side.) The lower limbs were moderately spastic, but handling, instead of increasing spasticity, seemed to produce flaccidity when the first resistance was overcome. The patellar tendon reflexes and the Achilles tendon reflexes were equal but exaggerated. There was no patellar clonus and no ankle clonus reflex. On irritation of the soles of the feet there was no movement of the toes. The patient had difficulty in controlling the sphincters of the bladder and anus.

Over the trunk tactile sensation was lost to a point as high up as if a line were drawn through the nipples. Sensations of heat, cold and pain were greatly diminished, but not entirely lost. The senses of touch, heat, pain and cold were lost throughout both lower extremities, but he rarely felt a pinprick over the anterior surface of the right thigh.

He complained constantly of severe pain in the lower extremities, and frequently asked the nurse to move his legs.

The Wassermann test of the blood appeared to be negative. Urine analysis showed evidences of cystitis. There was no deformity of the spine. The patient grew gradually weaker and weaker, and died, March 24, 1911, fifteen days after admission.

Postmortem Examination.—A fibromatous tumor was situated between the fifth and sixth cervical segments of the spinal cord. The tumor extended through the fourth cervical and fifth cervical intervertebral foramina, and grew about 2 inches downward into the soft tissues external to the vertebrae. The tumor had grown on the left anterior and left lateral surfaces of the cord, compressing the fifth cervical with the sixth cervical segment. The upper part of the sixth cervical segment was slightly compressed. The left fifth cervical anterior and posterior roots were involved by the tumor.

Examination by the Marchi method disclosed degeneration in one crossed pyramidal tract and in the posterior column of the same side. This was probably due to injury of a spinal root below the level of the lesion—evidently the descending fibers of the fifth and sixth cervical roots. The descending degeneration was surprisingly slight.

CASE 2.—*Fibroma of cervical portion of spinal cord beginning with pain in the right hand, with gradually increasing weakness of right upper limb. Four months later gradually and progressively increasing motor weakness of the left upper and both lower limbs.*

History.—Miss L. E. F., aged 35 (Lab. No. 400). The patient was healthy until 1897, when she first began to complain of numbness and a tingling sensation in the fingers of the right hand, associated with gradually increasing weakness, which caused her to drop objects. Several months later wasting of the muscles of the right hand was noticed. Gradually the whole arm became weak, and the pain extended to the right scapular region. Pain was much more frequent and severe at night than during the day. Several months after the numbness and tingling of the hand were first complained of the right lower limb grew weak, and the left upper limb also became affected. This condition increased steadily, so that after about four months the right lower limb was so weak that she was unable to walk without assistance. The left lower limb soon became similarly affected.

Physical Examination.—This revealed great weakness in the whole right and left upper limbs. The fingers and thumbs were habitually carried in flexion, suggesting the "claw-hand" appearance. The extensors were weaker than the flexors, although the latter had little power. The thenar and interossei muscles of each side were greatly atrophied. The right wrist reflexes were dis-

tinctly more marked than the left. Both lower limbs were weak and spastic, but the right was affected more than the left. The tendon reflexes in the lower limbs were both greatly exaggerated, the right more so than the left. Ankle clonus was absent on the left side, but was occasionally present on the right. Sensation was much diminished on the right side. (Notes incomplete.)

Constipation was present, an enema having to be administered every third day. Urine was passed involuntarily at times, but, as a rule, the patient was compelled to use a catheter.

Postmortem Examination.—A fibroma of the cervical region of the spinal cord was found.

Fig. 2 (Case 1).—Fibroma of the fifth and sixth cervical segments of the spinal cord that had caused gradually increasing weakness of all four extremities; severe pain in back and in legs. Upper limbs held in the Thorburn position.

CASE 3.—*Fibroma of cervical portion of spinal cord pressing on the first thoracic, eighth cervical, posterior spinal nerve roots that had caused severe pain in the upper and the lower limbs, followed three months later by slowly developing motor weakness.*

History.—Mrs. A. W. G., aged 43 (Lab. No. 539), was admitted to the service of Dr. Spiller at the University Hospital, Sept. 15, 1909. The patient stated that in October, 1908, she developed severe pain in the right hand. This

was diagnosed as neuritis. At this time she also had an extremely tender point in her back, on the left side, between the spinal column and the left scapula. The pain in her right arm continued for about ten weeks, and then gradually subsided and disappeared. Since that time she had had a persistent numb sensation in the fingers of the right hand. For some time past there had been pain in the left arm and numbness of the fingers of the left hand. About two and one-half months later—in December—the patient developed severe pain, limited to the left foot; this lasted about three weeks and then gradually disappeared. It was attributed to gout.

In February, 1909, about four months after the onset, she began to complain of a feeling of numbness about the waist, and of a sensation of distention in the abdomen.

About December, 1909, the patient first noticed that the left leg seemed to "lag" a little in walking, and in March, 1909, there was marked weakness in both lower limbs, although the left was weaker than the right. On March 27, 1909, while getting into a hot bath, she suddenly discovered that there were numbness and loss of sensation in all the parts below the waist. The following day the patient went to bed and remained there. After six weeks the weakness had increased to such an extent that, although just able to stand erect, she was unable to walk. She had now lost control of the bladder and rectum. Shortly after first going to bed she noticed that the legs would contract spasmodically. She stated that "any little thing" caused the muscles of the lower limbs to contract, and at the same time there would also be severe pain in the legs, radiating upward and stopping at the painful point between the shoulders. There was a constant burning sensation in the lower limbs, and the patient had a bed-sore of two or three weeks' duration.

Physical Examination.—The pupils were equal and reacted normally to light in accommodation and convergence. The movements of the eyeballs were normal. The cranial nerves all functioned normally. When the patient was turned to the right while lying down, there was sharp, stinging pain in the left middle finger. The grasp of each hand was normal. Biceps and triceps tendon reflexes were normal on each side, although the triceps reflex of each side may possibly have been less active than the biceps reflexes. Tactile, pain and temperature sensations were normal in each upper limb. No difference in sensation was apparent on the radial and ulnar sides of the limbs. Ataxia was absent, and there was no impairment in the sense of muscular position. Stereognostic perception was normal. The lower limbs were extremely rigid. The thighs were held flexed on the trunk, and the legs were strongly flexed on the thighs and could not be extended by the examiner. The patellar tendon reflexes could not be obtained because of the spasticity. Ankle clonus was present on each side, provided it was tested for when the spasm was partially relaxed. The Babinski sign was typical on each side. Tactile, pain and temperature sensations were lost throughout the lower limbs. There was pronounced involuntary jerking of the lower limbs. No deformity of the vertebrae, even at the point of tenderness in the back, was present. There was complete loss of control of the bladder and rectum.

September 20, an operation was performed by Dr. Frazier. The laminae of the first, second, third and fourth dorsal vertebrae were removed. On nicking the dura, cerebrospinal fluid spurted out for a distance of 2 or 3 inches, evidently under considerable pressure. About 50 c.c. of fluid escaped. After the cord had been fully exposed for a distance, including the first three thoracic vertebrae, nothing abnormal could be seen beyond a suspicious prominence at the level of the first thoracic segment. Because of the patient's condition,

further exploration was deemed inadvisable. The wound was closed with sutures. On the day following the operation the patient complained of almost constant severe pain, and died later.

Postmortem Examination.—A well-defined fibromatous tumor, measuring 2.5 by 1.7 cm., was found firmly attached to the anterior surface of the cord. The first left posterior thoracic root lay on top of the tumor, which pressed on and extended to the upper part of the first thoracic segment. The growth touched and pressed on the eighth cervical anterior root, but did not implicate it. The dura was adherent to the tumor in its upper portion. The growth was situated in front of the cord and did not extend to its right edge, but protruded beyond the left edge.

Fig. 3 (Case 2).—Fibroma of cervical portion of spinal cord beginning with pain in the right hand, with gradually increasing weakness of the right upper limb. Four months later gradually and progressively increasing motor weakness of left upper and both lower limbs.

Microscopic Examination.—Examination by the Marchi method showed intense degeneration of the ascending and descending tracts.

CASE 4.—*Fibroma of the thoracic portion of the spinal cord that had caused burning pain in the epigastrium for nearly three years before gradual development of spastic paraplegia and anesthesia; successfully removed by operation.*

History.—Fannie P., aged 49, was admitted to the University Hospital in Dr. Spiller's service, Oct. 8, 1920. The patient had entered the hospital in 1915 on account of burning pain in the epigastrium and vomiting, and had

left a few weeks later, against the advice of the staff. Pain had been attributed to cardiovascular disease. Examination at this time revealed no symptoms of disease other than evidences of beginning cardiac failure. She was readmitted to the hospital in October, 1920, still complaining of burning pain in the epigastrium, between the shoulders, and extending down the spine. She said the pain was severe at times. Examination showed that the tendon reflexes of the lower extremities were equal and much exaggerated. Ankle clonus was present on both sides. There was a positive Babinski sign on the right side, but none on the left. The spinal fluid showed 5 cells to the cubic millimeter; positive globulin reaction; sugar was present.

November, 1920, the muscular power of the lower limbs against resistance was fairly good for individual movements, but in walking her lower limbs appeared to be weak. The reflexes of the lower extremities were equal and exaggerated. Ankle clonus was distinctly marked on both sides. The Babinski reflex was absent on each side. Bone conduction, tested with the tuning-fork, was normal over both lower limbs. The sense of position in the toes was normal. Touch and pain sensations were normal over the lower limbs. The patient left the hospital before the examination could be completed.

She was readmitted in March, 1921, still complaining of severe burning pain in the back and in the epigastrium. On examination the following findings were noted: an exaggeration of the biceps and triceps reflexes; profound paralysis of both lower limbs, with exaggeration of reflexes, ankle clonus, and marked spasticity. Tactile, pain and temperature sensations were completely abolished below a point corresponding to about 2 inches above the level of the umbilicus. Vibratory sensation and the sense of muscular position were slightly impaired in the lower limbs. Spinal puncture elicited 4 c.c. of yellowish fluid; five cells per cubic millimeter; globulin positive. The Wassermann reaction was negative. Roentgen-ray examination showed no deformity of the spine. The patient suffered considerably from intermittent spasmodic flexor contractions of the lower limbs.

Treatment and Course.—April 12, 1921, an operation was performed by Dr. Frazier. The laminae of the fifth, sixth, seventh and eighth thoracic vertebrae were removed. A subdural tumor occupied the posterior aspect of the cord, extending from the eighth to the eleventh thoracic segments. The tumor extended farther on the left than on the right side, a small protuberance extending from the growth around the lateral aspect of the cord above the middle of the tumor. After the removal of the tumor the spinal cord was found to be very much flattened. Microscopic examination proved it to be a fibroma.

CASE 5.—*Intramedullary infiltrative tumor (ependymoma) of cervical portion of spinal cord; duration five years, causing slowly progressive increasing spastic paralysis of the limbs with marked muscular atrophy and increased reflexes and burning pain in the back.*

History.—Joseph M. (Lab. No. 671) was admitted to the University Hospital, Oct. 15, 1914, in the service of Dr. Spiller. In 1909 the patient first noticed weakness of the right arm, without pain. This progressed gradually. Two years later the right leg began to grow weak, and he had a sensation of numbness and tingling in the leg. Still later the left leg became similarly affected, and finally, in 1914, nearly five years after the onset, the left upper limb became paretic.

For the past three years the patient had had considerable burning pain between the shoulders and in the lumbar region, numbness and tingling of the lower extremities, with subacute aching pain in the knees and ankles.

Physical Examination.—This revealed marked kyphosis, beginning in the cervical region and extending into the thoracic region, with the point of greatest curvature at the fifth thoracic vertebrae. The ocular examination was negative. The cranial nerves were normal. The muscles of the upper extremities and shoulder girdles, including the supraspinati and infraspinati, deltoids, pectorals, and all the muscles of the upper arms, forearms and hands, were greatly wasted. The muscles of the thenar and hypothenar eminences were relatively well preserved. Those of the left upper limb were not quite so markedly affected as were those of the right upper limb; the latter was moderately spastic, and there was marked clonus at the wrist joint. Fibrillary tremors were present in the deltoids, biceps and triceps. The biceps and triceps

Fig. 4 (Case 3).—Fibroma of cervical portion of spinal cord pressing on the first thoracic, eighth cervical, posterior spinal nerve roots that had caused severe pain in the upper and the lower limbs, followed three months later by slowly developing motor weakness.

reflexes on the right side were exaggerated. The biceps on the left was exaggerated, but the left biceps reflex was absent. The muscles of the lower limbs were considerably wasted, and there was corresponding weakness. The tendon reflexes were equal but exaggerated, and there was abortive ankle clonus on each side. The Babinski reflex was also present on both sides. Cutaneous sensation of every form was impaired over the arms, trunk and lower limbs. At times the patient seemed to recognize pain, but his answers were uncertain, and exact limits were not determined.

Postmortem Examination.—This revealed marked fusiform swelling of the cord, its point of greatest intensity being at the lower cervical and upper thoracic segments. On cross-section, under the microscope, the specimen showed the characteristics of an ependymoma.

CASE 6.—*Sudden development of paraplegia caused by acute syphilitic softening of the spinal cord from thrombosis.*

History.—Robert McElreth (Lab. No. 267) was admitted to the Philadelphia Hospital, Dec. 20, 1904, in the service of Dr. Mills. After having been exposed to severe wet and cold weather in November, 1904, the patient was suddenly seized with "chills" and with a feeling of weakness, and was compelled to remain in bed. A few hours later he was unable to move his legs or to control his bladder and rectum. Vomiting occurred frequently.

Physical Examination.—This showed that the pupils were equal, but did not contract to light, although they did contract in accommodation. The cranial nerves were normal, as were also the upper limbs. The feet were swollen, deeply cyanotic and cold, pressure producing deep pitting. Both knee reflexes, although present, were decreased; no patellar reflex or ankle clonus was present, but there appeared to be a suggestion of dorsal flexion of the great toe. On plantar irritation he could move both legs slightly—the right more than the left. The man did not feel the prick of a pin below the knee, but at about that level, and above it, there appeared to be sensations to pain. Although oblivious to light touch on the thighs, he distinguished firm palpation, whereas over the abdomen and elsewhere sensation seemed to be normal.

Treatment and Course.—Dec. 20, 1904, 10 p. m.: The patient vomited, apparently the result of calomel. The bowels did not move. Temperature was lower; there was incontinence of urine.

Dec. 21, 1904: First sound just barely audible, about fifth interspace, in nipple line; second aortic sound could be heard faintly, but second pulmonic sound was not discernible. Breathing was better, easier and slower, and the pulse had become somewhat slower. The bowels opened by incontinence; there was a slight tendency to vomit after the midday meal.

Notes by Dr. McConnell: There was no palsy of any muscle supplied by the seventh or motor fifth nerve. The external ocular muscles were normal. The pupils were somewhat contracted, but equal; the left responded to light and accommodation; the right responded to accommodation but not to light. There was no loss of sensation to touch or pain over the face or scalp, and muscular power in both upper extremities was good. All normal movements were good. No wasting of the arm or forearm was apparent; the thenar eminence of the left side seemed somewhat less sound than on the right; there was no wasting of the interossei, although the first interosseous of the left seemed somewhat wasted compared to the right. Both upper limbs were the seat of numerous tumors. These were irregularly round, freely movable under the skin and not painful. They were somewhat soft, but were not cystic. The reflex was normal in the upper extremities of both sides. No tremor or fibrillary twitchings were present. All forms of sensation seemed to be well preserved.

Dec. 23, 1904: Following an enema the night before, the bowels moved well. The patient did not vomit, was perfectly clear mentally and fairly comfortable. At 9:30 this afternoon he suddenly complained of a sense of suffocation and died immediately.

CASE 7.—*Acute syphilitic myelomalacia of the thoracic portion of the spinal cord that had caused sudden onset of complete flaccid paraplegia and incontinence of urine and feces.*

History.—Peter S. (Lab. No. 787) was admitted Nov. 21, 1919, in the service of Dr. Spiller. In April, 1919, the patient first began to have difficulty in emptying the bladder. He was treated in another hospital, from which it was reported that a suprapubic cystotomy was done and the bladder drained. During operation the prostate was observed to be enlarged, but it was not removed. In spite of this difficulty the patient continued at his work until Nov. 11, 1919, when, after returning from work and feeling tired, he lay down on a sofa to rest. One hour later, on attempting to rise, he found that he was unable to do so. He stated that he felt dead from the waist down, and that he was unable to move either lower limb.

Fig. 5 (Case 4).—Intramedullary infiltrative tumor (ependymoma) of cervical portion of spinal cord; duration five years, causing slowly progressive increasing spastic paralysis of the limbs with marked muscular atrophy and increased reflexes and burning pain in the back.

Physical Examination.—The pupils were equal in size and normal, and reacted normally to light, accommodation and convergence. All cranial nerves functioned normally. There was complete motor and sensory paralysis of all parts below the level of the fifth rib on the left side and below the fourth

rib on the right side. Above this level no abnormalities were found. The arms were normal. The lower limbs were completely paralyzed and flaccid. The tendon reflexes were absent on each side, and all forms of sensation were abolished. There was incontinence of urine and feces.

Twenty-five c.c. of spinal fluid were withdrawn for examination and showed 3 lymphocytic cells to the cubic millimeter. The Wassermann reaction was not reported.

Roentgen-ray examination of the spinal column showed evidences of osteoarthritis of the sixth dorsal vertebra and the lumbar vertebrae.

The patient gradually grew weaker. The lungs became congested, and he was delirious at times. The patient died, Dec. 12, 1919.

Postmortem Examination.—Examination revealed complete softening of the spinal cord, involving the fifth, sixth, seventh and eighth thoracic segments. On cross-section it was impossible to differentiate between the white and the gray matter.

Microscopic Examination: The point of greatest intensity of softening was at the level of the fifth thoracic segment. There were thickening of the vessels and perivascular infiltration, but very little meningeal infiltration. Softening was diffuse, though greater in the posterior and lateral portions. Some of the vessels seen on cross-section appeared to be occluded.

Diagnosis: Syphilitic arteritis and meningomyelitis.

ABSTRACT OF DISCUSSION

Dr. Charles A. Elsberg of New York stated that he had always thought that the acute paralysis which occurs within a few hours or within twenty-four hours, as frequently seen in malignant disease of the spine, was due entirely to interference with the circulation of a localized area of the spinal cord and not to an acute myelitis from a toxic cause. When such spinal cords are examined postmortem or perchance are seen on the operating table, no evidence can be found of pressure on the cord, and the outer surface of the cord apparently looks normal.

Dr. Cadwalader deserved much credit for having called attention to the importance of the circulatory disturbances in these acute paralyses of spinal origin.

Dr. Elsberg stated that he hoped in the near future to give an explanation for early sensory pain symptoms and loss of motor power in the lower limbs in disease in the cervical cord. In spite of the evidence to the contrary, which is found in the literature, he believed that there is a lamellar arrangement in the various tracts in the pyramidal as well as in the spinothalamic pathways, and he hoped soon to have the clinical and experimental proof. For example, if it were a fact that in the spinothalamic tracts the fibers for the lower extremities coursed along the outermost parts of the tracts, it could easily be understood that a pressure from the outside would first affect the fibers for the lower extremities. In spite of all the evidence to the contrary, there was considerable evidence that a similar grouping occurred in the pyramidal tracts.

The probable reason that the symptoms which Dr. Cadwalader had described —that is, an affection of the lower extremities first in high lesions—were not more often observed was that a pressure is rarely so slow and gradual that the outermost portions of these pathways are first affected alone. Usually the entire tract is quickly involved.

Dr. Elsberg said that he realized and was fully aware of the fact that, up to the present time, the studies made of ascending and descending degenera-

tions in the spinal cord seemed to show that there is no grouping according to extremities of the fibers in the spinal tracts. But he believed that the evidence had been misinterpreted, and he hoped to be able to bring forward the proof of the correctness of his contention.

Dr. H. H. Hoppe of Cincinnati said that he wanted to add a contribution to the subject of the sudden development of paraplegia in lesions of the spinal cord running a subacute or chronic course.

A patient was admitted to the neurological service at the Cincinnati General Hospital with the history of pains affecting the abdominal region for a few months. Two or three days before admission, she developed weakness of the legs which advanced to a complete flaccid paraplegia in from twenty-four to thirty-six hours. At operation the dura was laid bare and a distinct loss of pulsation showed below the area of exposure. The pathologic condition proved to be a pachymeningitis interna hypertrophica. The lesion was located about the region of the eighth dorsal segment. The sudden development of the flaccid paraplegia in this case was in all probability caused by a compression of the anterior spinal artery.

Dr. William G. Spiller of Philadelphia said he was interested in Dr. Elsberg's remarks and awaited with much interest his presentation of proofs of a dissociation of the motor fibers for the upper and lower limbs in the crossed pyramidal tract of the spinal cord. All investigations, so far as he was aware, had shown that the fibers for the upper limb are not distinct from the fibers for the lower limb in the crossed pyramidal tract of the spinal cord. Experimental work on the motor cortex, in removing the center of the motor fibers of the upper limb or of those of the lower limb, has shown that the degeneration of the crossed pyramidal tract in the spinal cord as determined by the Marchi method is diffuse, and that the motor fibers of either upper or lower limb are not confined to a definite portion of the crossed pyramidal tract. It is doubtful whether such a separation of motor fibers for the limbs exists below the internal capsule, although theoretically one would expect a separation of this kind to exist.

In the posterior columns the fibers from the upper limb are distinct from those from the lower limb, for the column of Goll is believed to consist of fibers entering below the midthoracic region.

Dr. M. Allen Starr of New York said that he did not think all investigations pointed the other way and cited a case with illustrations, which was published in his book in 1902, of some very interesting specimens given him by Dr. Osler, in which a tumor lying opposite the first and second lumbar segments on one side resulted in an ascending degeneration of the posterior columns. This was traced upward at different levels and confirmed the statements that were published twenty-five years ago, that as demonstrated by the experimental lesions studies by the Marchi method there was a lamellar arrangement of fibers as they ascended the cord, each succeeding entering fiber lying on top and posterior to the fiber above it.

Dr. Henry M. Thomas of Baltimore said that he had been interested in a case which possibly might bear on Dr. Elsberg's remarks.

The patient was a school girl who had recently been operated on. Three years ago she had a pain in the left arm which was thought to be due to a toxic neuritis. She complained very little and went on with her school and athletic pursuits, but her hand became slightly atrophic. Three years after the onset she began to notice that she did not use her left leg as well as her right. This became evident about six weeks before the operation.

Her left hand was smaller than the right. She used it awkwardly, but it gave her little trouble. Her left leg showed the evidences of a mild pyramidal tract lesion. There was also a sensory loss of pain and temperature on the right side, involving the second thoracic segment and the whole side below. She had had no pain except that in her left arm three years before.

She was operated on by Dr. Dandy who found inside of the dura a small tumor, opposite the seventh cervical vertebra that hardly indented the spinal cord, but pressed on its left lateral aspect. This intradural tumor was an offshoot of a tumor the size of a lime that was in the body of the seventh cervical vertebra. He believed that the tumor was completely removed.

Dr. Thomas said that while the pressure on the spinal cord seemed to have been recent and slight, the sensory disturbance involved the whole side of the body, up to the level of the segment of the cord pressed on and did not involve the leg only, as might have been expected from Dr. Elsberg's experiences.

Dr. Lewis J. Pollock of Chicago said that it had been noted for some years that in cases of Brown-Séquard's paralysis, occurring as the result of lesions in the lower cervical or dorsal segments, analgesia was frequently absent in the lowermost sacral segments. It had likewise been noted that in patients recovering from this paralysis there was a caudal retreat of the analgesia. From these observations it had been concluded that there was a lamellar distribution of the ascending sensory tracts subserving pain and temperature senses. It had likewise been concluded that the fibers subserving the lowermost segments occupied a position lateral to those of the upper segments.

If this were true, there would be little difficulty in the differentiation of intramedullary from extramedullary lesions. When, in a case in which the lesion was situated in the dorsal or cervical segments, the sacral segments were not analgesic, an intramedullary condition was present.

Dr. Pollock had observed a number of cases of intramedullary lesions and found in them that the sacral segments were free from analgesia. However, in two cases of undoubted extramedullary lesions, one which at operation was found to be a pachymeningitis, these areas were likewise free from loss of pain sense. It seemed that such a condition could be explained more logically on the basis of a wide diffusion of the fibers from the lowermost segments as they ascend than on a lamellar distribution. It might be possible as there does not seem to be a crossing of the pain fibers below the twelfth dorsal segment, that in the lumbar and sacral portions of the cord pain sensation is transmitted through short fibers, as in the cat, in whom a bilateral hemisection of the cord at different levels fails to produce analgesia.

Dr. Charles A. Elsberg said, in answer to Dr. Thomas, that he had operated on several patients in whom tumors had grown through the dura, from the outside, and into the intradural space.

Recently he had witnessed an operation performed by Dr. Taylor, in which a tumor had evidently begun in one of the branches of a spinal nerve or near the spinal ganglion, had grown into the dural sac through the opening in the dura for the nerve root, and had then caused compression symptoms.

Dr. Williams B. Cadwalader of Philadelphia, in closing, said that he was glad his remarks had aroused some interest and thought that the views expressed by Dr. Elsberg regarding the arrangement of the sensory tracts within the spinal cord were most important.

A CASE OF FAMILY PERIODIC PARALYSIS*

M. NEUSTAEDTER, M.D., PH.D.
NEW YORK

Since comparatively few cases of this disease have been reported I feel justified in recording one recently seen.

REPORT OF A CASE

History.—J. M., a young man, aged 18½ years, a dental student, had a paternal cousin who died of cancer at the age of 30, his maternal grandmother died of diabetes, two maternal cousins were insane, three maternal uncles had periodic family paralysis, of whom one died in an attack at the age of 35, another died of pneumonia at 27, the third is living. He is 29 years old, and has had no attack for three years. The three uncles were not married. In two the attacks began at the age of 16 and in the third at 15. The one who died of pneumonia had the last attack about six months before his death. Otherwise there was nothing of importance in the family history. The parents could not tell whether cases had appeared in previous generations.

He is one of five children. The four are in perfect health. Since early childhood he stammered slightly. Otherwise the history was negative. He had never suffered from constipation.

His first attack was at the age of 14 years, during the evening. It was characterized by marked weakness in all extremities lasting a half an hour. He did not remember any exciting cause. The second seizure at 15, at night, completely paralyzed all extremities. In the third, eight months later, he became paralyzed in the evening and fully recovered in the morning. In the fourth attack, eight months later, he was paralyzed in the morning and did not recover until noontime. In the following one and one-half years he had five attacks at intervals of from three to six months, the last two at intervals of three weeks. All these attacks came on over night and lasted till afternoon. The paralysis was complete, involving all extremities, and the muscles of the trunk and neck. They were ushered in by a feeling of fatigue and numb ache in the extremities and palpitation of the heart, lasting at times a whole day. He would go to bed dizzy, sleep profoundly and in the morning be completely or partially paralyzed in the upper or lower extremities. The paralysis would grow progressively worse until all the musculature above mentioned would become involved. In some of the attacks he had difficulty in deglutition. On recovering, the muscles that became affected last would improve first, the distal ends of the extremities first, then the proximal parts. He invariably remained generally weak for twenty-four hours after a seizure. He vomited a number of times in all attacks, except in the last two. Headache was always present and perspiration was a constant accompaniment. It could not be ascertained whether the temperature was elevated during the attacks.

Present Seizure, February 1. The day before in college he was called on during the quiz hour for the first time. He became very much disturbed and was hardly able to walk to the rostrum. He managed, however, to answer the ques-

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*Read before the Section on Nervous and Mental Diseases at the Seventy-Second Annual Session of the American Medical Association, Boston, June, 1921.

tions correctly. On returning to his seat he felt the peculiar numb ache of the arms and palpitation, and at once "knew" that an attack was impending. This condition continued throughout the day and evening. His appetite was poor. He did his school work as usual and retired at the usual hour, 11 p. m., slept soundly and awakened at 6 a. m., paralyzed in the lower extremities and trunk. The muscles of the upper extremities soon became involved and then those of the neck. The face, speech and deglutition remained unimpaired. He perspired profusely.

At 12 m. the temperature by mouth was 100 F.; blood pressure, systolic, 80; diastolic, 60; pulse full and regular, 72; heart normal; respirations 24, fairly deep; the entire body moist.

Reflexes: The pupils were dilated, regular, equal, promptly responding to light and in convergence. The scleral, jaw, pharyngeal, biceps, triceps, radial, abdominal, cremasteric, Achilles and plantar reflexes were absent. The patellars were weak. There was no disturbance of sensation and no response to mechanical stimulation of the muscles. His physician reported that on a previous occasion no response was elicited by faradism.

Motor: The muscles of the neck were fairly strong; eye movements round; musculature of the shoulders flaccid and paralyzed; he was able to flex the forearm, hands and fingers as well as extend them weakly; pronation and supination were executed with some difficulty; respiration was normal, the intercostals and diaphragm were fairly active; the abdominal and dorsal muscles were paralyzed. At times he could flex the right thigh on the abdomen with great difficulty, but not the left. There was marked foot drop on both sides and no movement of feet nor toes. There was flaccid paralysis of the lower extremities. The consistency of his musculature presented no abnormalities. The mucous membranes, abdominal and thoracic viscera were normal. Fundi oculi were normal.

Intellect was intact. His appetite was poor. He passed urine several times; there was no sphincter disturbance.

At 3 p. m. the paralysis was gone; he could rise without difficulty and move all extremities. Motor power was fairly good, but failed after walking about thirty feet. The scleral, jaw, pharyngeal, biceps and radial reflexes were still absent; all others returned. He was still perspiring.

The chemical analysis of his blood by Dr. William C. Thro, showed blood sugar, 130 mg. per 100 c.c.; nonprotein nitrogen, 58.8 mg. per 100 c.c. of blood. The calcium of blood content could not be determined on account of the potassium oxalate, which was added to the blood as an anticoagulant.

Urine: Examination revealed: 1,550 c.c., collected during twenty-four hours; cloudy; yellow, acid; specific gravity, 1.025; indican normal; no albumin, sugar, casts, nor cells; amorphous phosphates and magnesium phosphate crystals present.

Nitrogen Excretion: The total nitrogen excretion was 1.223 per cent.; 18.9 gm. in twenty-four hours; urea nitrogen, 0.965 per cent., 79.0 per cent. of total nitrogen; ammonia nitrogen, 0.04 per cent., 3.2 per cent. of total nitrogen; purin nitrogen, 0.027 per cent., 2.2 per cent. of total nitrogen (+ uric acid); creatinin nitrogen, 0.024 per cent., 1.9 per cent. of total nitrogen; total creatinin nitrogen, 0.372 gm. in twenty-four hours; rest nitrogen, 0.167 per cent., 13.6 per cent. of total nitrogen; creatinin, 1.03 gm. in twenty-four hours.

The patient was put on a Schmidt diet for three days and the feces were examined. The following is the report: Mucus and blood absent; fermentation:

gas +++, reaction before neutral and after acid. Food residue: muscle fibers very few; small bits of connective tissue, no starch; parasites none, ova none. Bacteria: There were many fine gram-negative rods, very few *Bacilli aerogenes capsulati.* Urobilin normal. The feces show a carbohydrate fermentation type.

February 12, his blood calcium content was 20 mg. in 100 c.c., the highest calcium content ever reported. Oatmeal, milk, eggs and vegetables were eliminated from his diet for a month, and on March 12 his blood was again examined. This time the calcium content varied between 8.6 mg. and 9 mg. in 100 c.c. on repeated examinations. Nonprotein nitrogen now was only 39 mg. per 100 c.c. of blood; about 20 mg. less than on the day of the paralysis; blood sugar 166 mg. per 100 c.c., 36 mg. per 100 c.c. higher than before; creatinin, 1.2 mg. per 100 c.c.

In order to test the possibility of anaphylaxis he was vaccinated with seventy different proteins, consisting of food, epidermal and bacterial proteins, which the Arlington Chemical Company kindly placed at my disposal. All vaccinations proved negative.

March 12, he showed no physical abnormality whatever. There was, however, marked quantitative reduction to electrical stimulation with both currents as compared with another individual of the same age. His systolic blood pressure was 90, diastolic 60, pulse 78. There was a systolic murmur at the apex.

The differential blood count was: polymorphonuclears, 68 per cent.; transitionals, 10 per cent.; lymphocytes, 18 per cent.; large mononuclears, 1 per cent.; eosinophils, 3 per cent.; no basophils and no mast cells. The Wassermann reaction was negative.

April 23, Dr. Selian Neuhof examined the patient's cardiovascular system and reported "No evidence of cardiovascular disease. The abnormal systolic thrill at the apex and the split sound over the mitral area are of functional origin and due to cardiac hyperactivity. These abnormalities disappear almost entirely when the patient is lying down. The systolic blood pressure was at first 140; after a few minutes it fell to 118. This is evidence of heightened vasomotor tone. The diastolic blood pressure was 60. The orthodiascopic tracing shows a slender heart of graceful form, resting slightly on the diaphragm. This is assumed by some to fit in with the complex of vasomotor instability. The electrocardiogram shows normal rhythm. The deviations in the leads are normal in direction and size."

Treatment and Course.—June 4, he had two attacks. He was perfectly well on the previous day, and was sure there was no cause for worry or excitement. At 2 a. m., he woke up perspiring profusely, weak, and with a numb ache in all the muscles, dry throat and dizziness. Although he was not constipated, an enema was given at once, with good results. The weakness, however, progressed, and fifteen minutes after reaching bed he became totally paralyzed from the neck down. An hour later nausea set in, and he vomited three times during the day a fetid, greenish mucus.

At 10 a. m., his physical status was the same as in the last attack, with the exception that the temperature was 97 F. and the systolic blood pressure 140. The involved muscles responded to neither faradism nor galvanism. After a profound sleep, he woke at 3 p. m., fully recovered, but weak.

During the day he took 2 grains of calomel in fractional doses, followed by castor oil. Weak tea with milk was his diet the entire day. At 11 p. m. he vomited again and an hour later he was again completely paralyzed. He slept

soundly through the night, arose recovered but weak, and, June 6, was entirely well.

His blood, taken during the attack, showed a calcium content of 4.3 mg. per 100 c. c. The blood taken twenty-four hours after recovery contained 95 per cent. hemoglobin, 6,000 white cells, 40 per cent. polymorphonuclears, 5 per cent. transitionals, 38 per cent. lymphocytes, 10 per cent. large mononuclears, 5 per cent. eosinophils and 2 per cent. mast cells. The urine, 1,000 c. c. collected in the first twenty-four hours, had a specific gravity of 1.025; it contained a trace of albumin and marked increase of indican and a few cylindroids.

DISCUSSION [1]

The etiology of the disease is still obscure, but a psychopathic or neuropathic constitution seems to be the basis. This would be warranted by the familial character of the affection.

To Westphal's theory of a state of toxicity and Goldflam's autointoxication theory Schmidt adds the theory of ischemia, which, while transient in character, produces muscular changes. In such event a temporary lack of nutrition and transient accumulation of toxic materials may partially explain the syndrome. He further contends that an abnormally large amount of epinephrin is secreted in these neurotic persons and this produces the vasoconstriction. It is interesting to note that Orzechowski has produced typical paralysis in a patient suffering from periodic paralysis by injecting epinephrin. The reported glycosuria in some cases and the hyperglycemia in my case would point to a possible suprarenal glycemia.

If the theory of Schmidt is accepted, the specific toxin and autointoxication theories as a terminus ad quem and not a quo would fit into the genesis of the whole picture. The appearance of this syndrome in varying intensity at various ages, especially during and immediately after puberty, in most cases, and intermission during pregnancy may throw some light on this theory. The fact that disturbances of digestion begin and continue with the attack, characterized by nausea, vomiting, constipation, tenesmus, etc., excessive perspiration and also disturbances of heart action would certainly uphold such a theory.

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1. Full historical discussion of etiology and pathology of the disease will appear in the volume of transactions of the section on Nervous and Mental Diseases of the American Medical Association for 1921.

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ABSTRACT OF DISCUSSION

DR. E. W. TAYLOR, Boston: Dr. Neustaedter's investigations have added something to neurology. When this subject first attracted attention in Boston we arrived at no conclusion as to the etiology of this extraordinary condition. Dr. Neustaedter has evidently taken a definite step in the right direction, and I have no doubt some sort of definite chemical cause will be found for this paralysis, which stands unique in that during the attack the electrical reactions are entirely lost. It is a loss of muscular reaction, not muscle degeneration. The cases are so rare that they cannot be studied in bulk, but I hope when opportunity offers that Dr. Neustaedter will continue his work along the anaphylactic line of research.

News and Comment

SECOND ANNUAL MEETING OF THE ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

The second annual meeting of the Association for Research in Nervous and Mental Diseases will be held in New York during Christmas week. The program contains a symposium of research contributions on the etiology, symptomatology, incidence and pathologic characteristics of multiple sclerosis. Contributors were assigned special topics for investigation early in 1921.

The Commission will be furnished abstracts of papers beforehand, and, as at the last meeting, will put questions to the speakers on the material presented.

The proceedings will be edited and published in book form. The volume on epidemic encephalitis, the topic of investigation last year, is nearly completed and will soon be distributed.

The exact time and place of the December, 1921, meeting will be announced in a subsequent number of this journal.

Abstracts from Current Literature

REPORT OF A NUMBER OF OPERATED VERTEBRAL AND SPINAL CORD TUMORS. G. Mingazzini, Arch. f. Psychiat. **62**:121, 1920.

The writer's cases number six: two vertebral and four spinal tumors. Three of the latter were extramedullary. Two of the patients in the series (Nos. 3 and 4) had extradural echinococcus cysts and both made complete recoveries after surgical treatment.

Case 1 was a caudal perithelioma in a man aged 43, which lay beneath the third and fourth lumbar laminae. It was successfully removed surgically although the patient died some months later from marasmus. The symptoms were initiated by pain in the left lumbar region which extended later to the left nates and finally to the entire right lower extremity. Coincident with the pain in the right lower extremity, flaccid paralysis of the entire left extremity beginning in the left foot ensued, then the right foot became weak, also rectovesical symptoms developed. Examination of active motion showed only a slight extension of the left thigh possible; on the right side flexion of the leg on the thigh was distinctly weak. Riding breeches anesthesia of the sacral roots, hypesthesia of the lower lumbar roots to all kinds of superficial stimuli and loss of inferior tendon reflexes completed the syndrome.

In commenting on this case the writer calls particular attention to the segmental extension of motor paralysis which on the right side is explained as due to a compression of a spinal root contralateral to the tumor, thus differing from a cord lesion proper in which the transition is from monoplegia to complete paraplegia without segmental progression. This case shows that in caudal as well as conus lesions the disturbance of rectal and vesical function may be synchronous.

Case 2 was that of a vertebral sarcoma in a man aged 51 years, involving the lowest dorsal and the first two lumbar vertebrae. It ran a clinical course conveniently divided into three stages: The first stage consisted of lumbar pain and sudden loss of sexual power, separated by a pain-free interval from the second stage of aggravated and intense pain in the hips and the simultaneous loss of inferior tendon reflexes and onset of paraplegia accompanied by atrophy of the right quadriceps. The third stage was marked by retention of urine, pressure pain over the first two lumbar vertebrae and edema of the lumbar spine. The only sensory disturbance present was a band of hypesthesia in the upper and posterior portion of the left thigh for all kinds of superficial stimuli. Active motor function was conserved in the legs and feet, but considerably impaired in the thighs which could neither be completely flexed, abducted or extended. Adduction was possible. Lumbar puncture revealed a fluid of yellow color (xanthochromia) which coagulated spontaneously and also contained an excess of leukocytes. Roentgenograms of the spine were negative. The presence of a positive tuberculin reaction suggested tuberculosis, which, however, was eliminated from the clinical picture. In favor of a vertebral rather than a caudal tumor was the marked rigidity of the trunk and the slight disturbance of the objective sensibility as contrasted with the marked subjective sensibility disturbances.

An attempt at operative relief resulted in determining the character and site of the lesion, which, however, did not involve the dura or interrupt the integrity of the cord or spinal roots. The patient died from cardiac failure at the close of the operation.

The author does not recall in the literature such an edema as he describes, although he quotes Oppenheim and also Bruns as mentioning a widening of neighborhood cutaneous veins in such cases. As an explanation it is suggested that the blocking of the lumbar efferent veins at the intervertebral foraminae may be the causative factor. From the radiographic standpoint this case is quoted in support of the contention of Flatau that the roentgen rays offer little aid in the diagnosis of vertebral tumors. This has been quite opposite to the experience of the reviewer, especially in this region of the spine.

In Case 3, the patient, a woman aged 40, complained first of left lumbar pain radiating into the left hypochondrium, then weakness developed in the left lower extremity rapidly extending to the right lower extremity, finally rendering the patient completely paraplegic. Both patellar reflexes were exaggerated and clonic contractions in the lower extremities, particularly the left lower extremity, were observed. Sensory disturbances reached as high as lumbar 2-4. Three periods of the clinical course were distinguished: neuralgic pain, Brown-Séquard syndrome and paraplegia. The course of the affection was about ten months. Tuberculosis of the spine was first suspected because of a swelling to the left of the spine in the region of the last two ribs. Roentgenograms were, however, negative and it was only by repeated exploratory punctures, the last revealing a gelatinous yellowish fluid containing daughter cysts, that the diagnosis was established. The operation disclosed a cord compression by the cyst which had eroded the arches of the last two dorsal and first lumbar vertebrae but did not perforate the dura.

In Case 4 an echinococcus cyst of the lung had been extirpated eight years before. Pain in the right lower extremity of a sciatica-like character and distribution was the first symptom. Five months after the onset of pain a swelling was noticed at the fifth lumbar vertebra, and this eventually proved to be a recurrence of the echinococcus infection. The neurologic examination at this stage showed absent Achilles' reflexes, hypotonicity of the right calf muscles and a striking weakness in the flexors of the right thigh on the pelvis. Neither atrophies nor objective sensibility disturbances existed. The cyst was found to extend into the last two lumbar vertebrae.

The fifth case reported was that of a woman 45 years old, who five years previously had her uterus removed because of a myoma. An intradural fibrosarcoma compressed the cord at the level of the seventh cervical segment producing a Brown-Séquard syndrome. About a year before coming under medical observation paresthesias were first noticed in the region of the right scapula and somewhat later were felt in the right upper extremity, radiating to the first three fingers. Some eight months after the onset of these paresthesias pain was felt in the right upper extremity, and occasionally in the left upper extremity, in the back of the neck and in the upper part of the sternum. Then paresthesias in the left lower extremity developed and later were felt in the left side of the abdomen. Weakness of the right hand and of the right lower extremity was complained of.

Examination showed a hypotonia of the musculature of the right upper extremity with definite atrophy of the hypothenar eminence and marked loss of strength, especially in the forearm and hand. In the lower extremities drop foot and outward rotation was found on the right side, with considerable

weakness in all three segments of this extremity, with especial lack of tone in the right calf muscles. The tendon reflexes on the right were increased over those on the left, and ankle clonus and the Babinski reflex were both present. In the left lower extremity and abdomen marked disturbance of painful and temperature sensation was found. At higher sensory levels the ulnar border of the right hand and forearm presented at times varying degrees of hyperesthesia and hypesthesia; bilaterally in the posterior cervical region a hyperalgesia existed. From the standpoint of the deep sensibility, astereognosis and impaired weight discrimination was found in the right hand, and in the left lower extremity hyperpallanesthesia and parapallanesthesia. The case therefore presented a Brown-Séquard syndrome. The cerebrospinal fluid was not remarkable. The pulse was ordinarily somewhat slow (60) and in the last month of observation a slight elevation of temperature was noted (37.8 C.). The spine was held upright and stiff.

The tumor, 3.5 cm. long, was found beneath the dura by removing the sixth and seventh cervical spines. It was seen to compress the right posterior columns of the cord and the eighth cervical root. The recovery was uneventful, only an atrophy of the right hypothenar eminence remaining.

In the analysis of this case the variations of the subjective disturbances of sensibility illustrated the impossibility of early localization of the tumor by this means. The disturbance of vibration sense due to irritative causes corroborates Frank's postulation that the fibers which transmit vibration ascend homolaterally in the region of the lateral pyramidal tract, and that pallanesthesia is even a more constant symptom in pyramidal tract lesions than the Babinski sign.

The last case of the series, that of a woman aged 29, was similar to the foregoing case in that the cord was compressed by an intradural fibrosarcoma located beneath the fifth, sixth and seventh dorsal spines, producing a Brown-Séquard syndrome. The clinical course could be divided into three stages, the first of radicular pain corresponding to the first and second lumbar roots on the left side. Second Stage: A Brown-Séquard paralysis (objective sensibility disturbance in the right lower extremity and paresis of the opposite extremity). The objective sensory disturbances affected touch, pain and temperature and extended as high as dorsal 9. The motor disturbances in the left leg at this stage were predominately flexor paresis, as flexion of the thigh was only possible to half the normal range. Extension of the leg on the thigh was possible, but not flexion. Limited flexion and extension of the foot was possible, but not adduction or abduction. The left tendon reflexes were exaggerated. Third Stage: Final paralysis of the extensors of the left thigh and complete paralysis below, with bilateral ankle clonus and Babinski sign. Pallanesthesia and bathyanesthesia were present on the left side. Difficulty was experienced in emptying the bladder and in retaining feces.

Although the tumor was entirely removed at operation there remained a spastic paralysis in flexion on the left side or side of the motor paralysis. This was attributed to the long duration of the compression (three and one-half years) as contrasted with the much shorter duration of the previous case.

SCHALLER, San Francisco.

TUBERCULOTOXIC MENINGITIS. HEINRICH HERSCHMANN, Arch. f. Psychiat. u. Nervenkrankh. **62**:3, 1921.

In the clinic for tuberculosis there are many complaints of headache which always causes the staff to be on the lookout for a beginning tuberculous men-

ingitis. In some patients the headaches disappear, and these cases are classed as meningeal irritation or meningismus, without any distinction from tuberculous meningitis and without an investigation into their pathology. Since Quincke, a toxic form of serous meningitis in infections in other parts of the body, such as those in typhus and pneumonia, is well known. The author has written an article concerning meningitis in dysentery.

With regard to the mechanism of the tuberculotoxic effect on the nervous system, there are only meager references in the literature, and on most points there is much controversy. This is discussed in French literature by Laignel-Lavastine and Armand-Delille. The former found in tuberculous meningitis changes in the cells of the cortex which were considered to be independent of the meningovascular processes. The involved cells showed the characteristic signs caused by poisoning. He means by this that the cell changes through the poison of tuberculosis are like those of other poisons of infectious diseases, such as pneumonia. Here Oppenheim gives the anatomic confirmation by stating that the poison can cause direct cerebral symptoms.

The possibility of a direct injury to the meninges by the tuberculosis toxin is up to the present doubtful and a search through the literature revealed the following case of Riebold: A 14-year old boy with bone tuberculosis became ill with meningitic symptoms which disappeared in three days. Four weeks later he developed similar symptoms from which he died very soon. In the discussion the author suggests that at first there was a serous meningitis produced by the tuberculosis toxin and the symptoms quickly cleared up, but this had formed a favorable place for the lodging of the tubercle bacillus. More convincing is the work of Villaret and Tixier who have a more authentic case of tuberculous meningitis in which search was made for the tubercle bacillus, eventually by direct study (stain in sections) and also by animal inoculation, but the germs were not found. They conclude that in this case the process was the result of the toxin. Dr. Herschmann then quotes his own case: A young man, 21 years old, seen Feb. 21, 1919, complained of severe left-sided headache which had been present since November, associated with optical sensations in the form of light zig-zag figures like lightning. Recently he had suffered from loss of appetite, night sweats and a slight cough, but he had had no fever and no brain symptoms. At this time the examination was negative except that the roentgenogram undoubtedly showed intracranial pressure, and there was active tuberculosis of the left apex. Lumbar puncture revealed no pellicle; the globulin was increased; there were 400 cells, of which 90 per cent. were lymphocytes and 10 per cent. polymorphonuclears. No bacteria were found and animal inoculation was negative. Gradually the cell count came down, and the final puncture showed a normal count. He was discharged April 25. In the résumé the author states that it is certain that this patient had a meningitis as shown by the enormous lymphocytosis of the spinal fluid. In contradistinction to the intensely positive findings in the spinal fluid is the poverty of clinical symptoms. Remarkable is the fact that the roentgen ray showed the increase of intracranial pressure three months after the onset. Of great importance is the question of how this peculiar subacute meningeal process had originated. There was no trauma, no disease of the ears and sufficient study showed only tuberculosis of the lungs. The diagnosis of tuberculous meningitis is not verified, but the author quotes Cushing who states that while the lumbar puncture fluid may be sterile, the ventricular fluid may contain the germs, and Foerster's statement that in localized tuberculous meningitis frequently no germs are found in the spinal

fluid. The second case quoted is that of a man 51 years old who had suffered for a long time from tuberculosis of the lung. He began to show mental confusion with various delusions, etc. Two children died of tuberculosis. During the last month he complained of headache and was confined to bed for two weeks. On admission he was disoriented. He complained of headache. Examination of the lungs showed a cavernous tuberculosis of both lungs. A few days later he felt pain on pressure over both eyes; the lower extremities were slightly spastic with a Kernig sign on both sides; there was generalized weakness but no paralysis. Otherwise he was normal. Lumbar puncture showed that the spinal fluid was not under pressure and that there was no discoloration. After standing twenty-four hours there was a flocculent precipitate. The Wassermann reaction was negative. There were 43 lymphocytes and 4 polymorphonuclears to each cubic millimeter. The globulin reaction was positive. Bacteriologic study was negative. The patient died of pneumonia a few days later. The microscopic findings in the brain showed that the pia everywhere was considerably thickened—up to from eight to ten times its usual thickness. There were in other parts of the pia portions which were poor in nuclei and dense; there was vessel-free connective tissue which looked like degenerated hyaline tissue. On the inner side there was rich, young edematous connective tissue. The tissue of this zone was filled with blood and greatly infiltrated with cells—in part round, lymphocytic nuclei and in greater part oval or spindle-shaped connective tissue nuclei. The cell forms did not show the characteristic arrangement of tubercles; epithelioid and giant cells were not seen. In commenting on this case, the author states that without the necropsy findings one would consider this a beginning tuberculous meningitis, but the histologic findings show it to be an acute exacerbation of a chronic meningitis.

In conclusion the author states that the findings of Villaret and Tixier, the cases of Riebold and Busse, likewise his own cases, make the existence of a tuberculotoxic meningitis probable.

WINKELMAN, Philadelphia.

A CONTRIBUTION TO THE PATHOLOGY OF THE HYPOPHYSIS.

VON MONAKOW, Schweiz. Arch. Neurol. u. Psychiat. **8**:200-207, 1921.

The rôle that the hypophysis plays in producing the condition of Fröhlich's syndrome is to this day not entirely clear. Whether the anterior lobe participates, as Cushing thinks, the pars intermedia, as Biedl thinks, or the neurohypophysis, as B. Fischer thinks, or even whether the hypophysis is at all responsible, is still a mooted question. While we are familiar with the fact that a puncture of several portions of the nervous system will produce metabolic disorders, for instance glycosuria, it must be remembered that we are quite unfamiliar with what portion of the nervous system is injured; whether cells or tracts are irritated, or whether they are destroyed. The relationship of these phenomena to the suprarenals and the liver is, furthermore, unknown.

Von Monakow reports the case of a man, 58 years of age, in whom there was almost complete disappearance of every portion of the hypophysis, with the exception of the posterior lobe, which was practically unchanged. The pathogenesis of this lesion was not clear. It is possible that it was secondary to a slight internal hydrocephalus, although the patient had never given any clinical evidence of this disorder, and the necropsy findings were very slight. There was no evidence of tumor anywhere.

The clinical course was as follows: The patient was first seen four years previously, at which time he was suffering with an acute articular infection of unknown etiology. It was noted at the time that his skin was a little dry and atrophic; the testicles were slightly diminished in size; there was a slightly active apical tuberculosis, slight weakness of the facial muscles and increase of the right patellar reflex. Four years later the patient showed marked changes. There was complete disappearance of all hair except that on the head; the skin was dry, thin and alabaster in color; the testicles were extremely atrophic and on section showed not only disappearance of the seminiferous tubules and absence of all spermatozoa, but also complete obliteration of the interstitial cells of Leydig; the penis and prostate were practically unchanged; there was a marked disproportion between the amount of fat and the actively tuberculous process, which was responsible for the fatal termination through peritoneal involvement. Although there was a daily fever and the patient took comparatively little food, he lost scarcely any in body weight, and the panniculus adiposus was everywhere well developed and presented an eunuchoid distribution. Examination of the urine was practically negative; blood chemistry normal; 3,200,000 red cells, hemoglobin 55 per cent.; blood pressure normal; the Wassermann test was negative. It may be added that the fundus examination and roentgenogram of the sella were normal. The administration of 150 gm. of glucose and the injection of 1 mg. of epinephrin caused no glycosuria. Blood pressure, also, was not altered. The administration of thyroid extract did not change the pulse rate, blood pressure or the amount of urine. Pituitary extract had not the slightest effect.

There were two factors of great interest. The first was the peculiar psychic attitude, which was not dependent on any structural nervous lesion of an ordinary character. The patient was markedly apathetic; sexual desire, which had been normal, there having been five healthy children, had diminished for the past six years and was now completely absent. He became indifferent and somewhat stupid; he reacted little to his environment and to his own situation; he never complained; he had no appetite and never called for fluids. His apathy was so marked that he made no attempts to brush away flies, which at times completely covered his face. Answers to questions, however, were always clear and relevant. There was marked physical weakness, which could not be explained on the basis of tuberculosis or the general condition of the muscles.

The second point of interest was the urine output. The hypophysis is looked on by some as giving rise to a hormone that influences the kidney in an inhibitory manner. The disappearance of this hormone, which is supposed to rise from the pars intermedia, should produce a diabetes insipidus. While the pars intermedia was completely absent, he at no time had polyuria; on the other hand he was oliguric, excreting about from 400 to 500 c.c. of a specific gravity of 1.016 to 1.018 daily, a phenomenon that ran parallel to the general slowing down of all vegetative functions. Only shortly before death was there an albuminuria and cylinders and fat granules were found in the urine. At necropsy marked degenerative changes were noted in the kidneys, indicating the presence of a nephrosis. It would appear, from experimental work as well, that there is a relation not only between the hypophysis and other endocrine glands, but also between hypophysis and the kidney, although this point of view must still be considered purely hypothetical.

WOLTMAN, Rochester, Minn.

COMPRESSION SYNDROME IN CEREBROSPINAL FLUID WITH SPECIAL REFERENCE TO THE QUECKENSTEDT SYMPTOM. KARL CROSZ and MARTIN PAPPENHEIM, Deutsch. Ztschr. f. Nervenh. **67**: 1921.

A voluminous literature has developed on the so-called compression syndrome in the cerebrospinal fluid since Nonne, in 1908, first drew attention to its diagnostic importance. Albumin has been noted in great quantities. In a case of von Reichmann's it reached 4.6 per cent. At times there is spontaneous coagulation and in some instances xanthochromia. The syndrome is found in extradural, intradural and even in intramedullary tumors, in the latter cases being probably brought about through a collateral edema. It has also been found in cases showing tubercles, spondylitis, cysts, echinococcus (Queckenstedt), cysticercus (Goldstein), meningitis serosa and serofibrinosa circumscripta, funicular myelitis and in some cases in which diffuse disseminated necrotic changes have occurred, as in different forms of subacute chronic meningitis. The syndrome, namely, high albumin content with normal or slightly increased cell count and at times spontaneous coagulation and xanthochromia, is diagnostic of some obstructive process in the subdural space.

Froin and Ledoux report a case of gumma with meningitic adhesions (necropsy), the fluid of which showed xanthochromia and spontaneous coagulation.

Queckenstedt described a symptom present in cases of obstruction, namely, that pressure on the veins of the neck either failed altogether to produce a rise in the fluid pressure after lumbar puncture or at any rate such rise was delayed and insignificant, in marked contrast to the rapid rise seen in normal cases. He reports five such cases. Later von Redlich described a case of tumor with a positive von Queckenstedt symptom, and von Pappenheim reported it in a case of tuberculous meningitis. Others have also reported this symptom.

The authors' series consists of 14 cases showing symptoms of obstruction, 8 of which were diagnosed as tumors of the dorsal portion of the spinal cord, 1 a tumor of the lumbar cord demonstrated at operation, 1 a cervical tumor (necropsy), and 4 cases of vertebral caries—2 cervical and 2 thoracic—3 of which were confirmed at necropsy. In 2 of the tumor cases there was a slight cell increase of 12 and 13 cells per cubic millimeter. The spinal fluid pressure was measured and Queckenstedt's test was performed in 9 cases, namely, in 4 cord tumors of the dorsal region (3 verified), 1 cervical cord tumor, and in all 4 cases of vertebral caries. The Queckenstedt symptom in the 4 cases of caries and in the 3 verified tumors was positive. In 1 case of a tumor of the eleventh thoracic vertebra in which the albumin content of the fluid was normal, it was the only positive symptom. In another case with a Brown-Séquard syndrome (D 6 — D 7) it was only faintly positive. In a case of paraparesis a positive Queckenstedt symptom became negative after a small amount of fluid had been withdrawn.

The fluid pressure in a few cases was high. In one case of caries of the dorsal region it registered 220 mm. of water and in two cases of tumor 240 mm. and 260 mm. respectively. In the others it was normal. In the foregoing case of caries the pressure dropped rapidly from 220 mm. to 110 mm. after 5 c.c. of fluid had been withdrawn. There is no explanation given for the increased pressure in these cases of obstruction; however it was noted that in two of the three cases with increased pressure the obstruction was not complete.

An increase in globulin and total albumin as determined by the Ross-Ione and the Nonne-Appelt and Pandy methods was noted in twelve cases. In general, the total albumin was not over 2 per cent., but in one case of cervical caries it amounted to 4 per cent. In the tumor of the lumbar cord there was a total albumin content of 1 per cent. with spontaneous coagulation, and this was the only case in the series which showed xanthochromia. In the literature xanthochromia has been reported with relatively greater frequency, and varied explanations have been offered concerning its nature and cause. It is generally conceded, however, to be a hemoglobin derivative gaining access to the fluid either through actual hemorrhage or by passage through the undamaged vessel walls.

The colloidal gold reaction in the patient with xanthochromia and in the patient with caries with great increase in albumin content showed a meningitic curve.

In a case of cervical caries fluid taken during operation from above the obstruction proved to have a normal albumin content which was in marked contrast to the fluid from below the lesion. Others have made similar observations.

The fact that cases of brain tumor almost regularly show similar spinal fluid changes to the ones described render these tests of no differential value. The authors report seven of a series of eight brain tumors showing a moderate increase in albumin of from 0.5 to 1.5 per cent.

The French lay emphasis on a syndrome consisting of increased albumin content with spontaneous coagulation and xanthochromia, first described in 1903 by Froin. At first, hemorrhagic processes were thought to be the cause, later it was deemed to be due to low pressure in the sac which caused changes in the vessel walls and allowed exudation. By some it was considered to be a result of fluid stasis. Baruch assumes that the tumor itself secretes an albuminous fluid.

Regarding the nature of the compression the French give three possibilities: pressure from without, fibrous, meningomedullary adhesions, and acute inflammatory conditions.

In conclusion, the authors add five cases as further proof that the compression syndrome when found in inflammatory conditions may not be due solely to this pathologic process but also to an actual obstruction as demonstrated by a positive von Queckenstedt symptom. Case 1: Tuberculous meningitis. The pressure was 220 mm. which dropped to 70 mm. after 6 c c. of fluid had been drained off; positive Queckenstedt symptom, xanthochromia, 70 cells per cubic millimeter, 40 per cent. of which were polymorphonuclears, strong phase 1. The next day the pressure was 180 mm., which dropped to 0 after 5 c.c. of fluid had been withdrawn. There was marked xanthochromia, 60 cells per cubic millimeter, total albumin almost 2 per cent., with spontaneous coagulation. Necropsy showed extensive exudative meningitis. Case 2: Epidemic meningitis showing light colored fluid, pressure 100 mm., positive Queckenstedt symptom. Following the removal of 2.5 c.c. of fluid the pressure dropped to 60 mm. There were 700 cells, 60 per cent. of which were polymorphonuclears. Strong phase 1, albumin content about 6 per cent. Case 3: Purulent meningitis (following injury); turbid fluid, pressure 220 mm., which dropped to 170 following the withdrawal of 3.5 c.c. of fluid, positive Queckenstedt symptom, several thousand cells chiefly of the polymorphonuclear variety; albumin about 5.25 per cent. Case 4: Positive Queckenstedt symptom in a case showing localizing symptoms in the lower thoracic region. The patient

admitted syphilitic infection nine years previously. At present the blood Wassermann reaction was negative, but the spinal fluid was +++. The fluid pressure was 60 mm. which dropped to 0 on the withdrawal of 3 c.c. of fluid; globulin and albumin increase 3 per cent.; 185 cells per cubic millimeter; no xanthochromia. The patient improved under treatment. Case 5: First seen by von Muller who noted at operation inflammatory changes with adhesions of the cord membranes. The authors later performed a spinal puncture on the same patient and noted a normal fluid pressure, a markedly positive Queckenstedt symptom, 16 cells per cubic millimeter, and slight increase in globulin and albumin.

NAGEL, San Francisco.

BASEDOW'S SYNDROME WITH TROPHÈDEMA. C. J. PARHON and A. STOCKER, Rev. neurol. **27**:1020 (Oct.) 1920.

The association of hyperthyroidism with trophedema impresses the authors as of sufficient significance to justify report, particularly so since the mention of twenty-five cases in the literature to date indicates the possibility of more than merely coincidental association between the two conditions.

Their case was that of a girl 21 years of age. The family history was negative, with the exception of alcoholism in one grandfather, tuberculosis in one brother and the existence of a psychosis (type unspecified) in a paternal step-brother. The personal history was negative until the beginning of the present trouble except that during childhood she had diphtheria, whooping cough, measles and malaria. The present trouble dated back to a severe affective shock sustained two years previously as a result of a "Bolshevik" raid in which a member of the patient's party was murdered, the patient, herself barely escaping with her life. This strain was definitely aggravated soon after by the necessity of testifying at the trial of the guilty parties. As a result, the patient was profoundly prostrated and in addition to general physical and nervous weakness there were noted crying spells, irritability, cardiac distress, vomiting and insomnia. After some time the thyroid was observed to enlarge, particularly on the right, and there was marked protrusion of the eyeballs. The patient also complained of headaches and distressing pulsation in the right arm and right side of the face. There was also thinning and lightening of the hair and eyebrows. Some time later the patient noted a swelling about the ankles, which ultimately involved both legs. There was cessation of menses for two years, from the time of the accident until the patient entered the authors' service.

On examination the patient complained of marked weakness and ready fatigability and showed marked exophthalmos, Dalrymple sign, marked thyroid hypertrophy, particularly of the right lobe and the isthmus, thinning and lightening in color of the hair and eyebrows, somewhat accelerated respiration (22) accompanied by a feeling of thoracic oppression and occasional dyspnea. The heart rate was 120, and there was complaint of constant distressing palpitation. The extended fingers showed marked tremor. The legs showed increased circumference to a level just above the knees and presented a somewhat edematous appearance, although there was no evidence of pitting following pressure nor any indication of temperature or color change. The condition seemed essentially a uniform subcutaneous or areolar fatty infiltration, and suggested itself to the author as typically trophedema. As a result of this condition the feet were held in a slightly extended position and

flexion movements seemed somewhat difficult. The patient complained of ready fatigue on movement and was able to change from one position to another only after great effort. She complained of a feeling of heaviness in the legs and pain in the tibio-tarsal articulations, which was definitely accentuated on movement. There was no change in reflex response except that the knee jerks seemed somewhat diminished. Differential blood count showed only 58.5 per cent. polymorphonuclears of which 5.2 per cent. were eosinophils. The mononuclear cells were mainly of the large type.

In discussing this association of hyperthyroidism and trophedema, on the basis of their own case and those described in the literature, Parhon and Stocker feel that the latter, mainly because of its segmental nature, was induced essentially as a result of vegetative nervous system disturbance, probably intramedullary in locus, which was determined primarily by the existing endocrine imbalance or dystrophy.

RAPHAEL, Ann Arbor, Mich.

COMPRESSION SYNDROME OF THE CEREBROSPINAL FLUID IN TUBERCULOUS SPONDYLITIS. W. RAVENS, Deutsch. Ztschr. f. Nervenh. **68**:69, 1921.

The author begins by stating that he has previously been able to report a few cases of tuberculous spondylitis in which the spinal fluid showed a positive phase, one without cell increase, with at times xanthochromia. Among others, Henkel, Schwartz, Bremer, Queckenstedt and Zaloziecki have reported similar findings. Sicard and Foix report nine cases of dorsolumbar pachymeningitis following Pott's disease, in which the spinal fluids were of light straw color, with increased albumin content but no increase in cells. Schnitzler found a positive Phase 1 in a suspected case of cord tumor which proved to be vertebral caries at operation. This led him to make a fluid examination in seven other cases of Pott's disease. Three of these presenting symptoms of cord pressure showed a typical so-called compression fluid, namely, high albumin content with normal cell count, while four uncomplicated cases had normal fluids.

The results of spinal puncture in forty-one personally observed cases of Pott's disease are reported. Twenty-seven patients were male and fourteen female; twenty-five were between the ages of 3 and 10 years; eleven from 11 to 20 years, and five from 20 to 42 years. The disease affected the cervical region in two, the thoracic in twenty-seven, and the lumbar region in twelve instances. The danger of puncture was not considered great in these cases and no ill results are reported.

Twenty-three of the patients showed no cord symptoms and had normal fluids, fifteen had distinct changes in the fluid and three had marked changes. All of the latter eighteen cases had more or less marked symptoms of cord pressure varying from a complete spastic paraplegia with bladder and rectal symptoms to minor local sensory disturbances. In all these eighteen cases the spinal fluid was clear, colorless, with normal or increased pressure. There was a definitely positive Phase 1 in all and it was marked in three cases. There was no cell increase in any of the forty-one cases.

The author concludes that fluid changes in certain cases of spondylitis do not differ qualitatively or quantitatively from those found in tumors which cause obstruction to the flow of the fluid. This fact is considered of more theoretic than practical interest.

The main points of the article are thus summarized:

1. In tuberculous spondylitis the same fluid changes may occur as in tumors compressing the cord.

2. These changes occur in those cases in which there has been pressure on the cord.

3. This fact will not greatly increase the difficulty of differential diagnosis.

4. The degree of increase in albumin runs parallel with the severity of the nervous symptoms.

5. With improvement in clinical symptoms the total albumin content diminishes.

NAGEL, San Francisco.

THE STATIC OR POSTURE SYSTEM AND ITS RELATION TO POSTURAL HYPERTONIC STATES OF THE SKELETAL MUSCLES. J. RAMSAY HUNT, Neurol. Bull. 3: No. 6 (June) 1921.

In discussing a case of decerebrate rigidity, Hunt reviews his conception of the static and kinetic systems, the various disturbances of motility and posture and considers the different forms of hypertonicity. The following is a brief summary of his views, which he has already stated on previous occasions.

The kinetic system concerns itself with active movement, the static with passive movement or posture. Both active motility and posture may be divided into reflex, automatic-associated and isolated-synergic or dissociated. These types of movement may be correlated anatomically with the reflex or segmental nervous system, with the paleo-encephalic (corpus striatum-optic thalamus) and neo-encephalic (cortical) mechanisms for the kinetic system and the paleocerebellum (vermis, its nuclei and pathways) and neocerebellum (celebellar hemispheres) for the paleostatic and neostatic systems. The paleokinetic and neokinetic systems are subserved, respectively, by the pallidal and pyramidal systems. The afferent pathways for the static system are by way of the corticopontocerebellar and spinocerebellar pathways while the efferent pathways of the neostatic and paleostatic systems, respectively, are by way of the superior peduncle to the nucleus parvicellulatus and nucleus magnocellulatus of the nucleus ruber. Hunt postulates the existence of distinct efferent fibers in the peripheral nerves and separate endings in the muscle spindles, each subserving the static and kinetic systems, and he points to the sarcoplasm and anisotropic disks and the demonstration of nonmedulated fibers in the peripheral nerves to prove his conception of the double motor innervation, thus showing the existence of two systems all through the nervous system even to the final motor end plates. Without going into too many critical details, it may be pointed out parenthetically that the demonstration of nonmedullated nerve fibers and distinct end plates, which speak for double motor innervation, has been shown to refer not to the static or postural mechanism which Hunt conceives of but to the sympathetic nervous system. (See the recent article by J. T. Wilson on the "Double Innervation of Striated Muscle," *Brain* **44**: Pt. 2, 1921.)

Based on the foregoing anatomic-physiologic considerations, Hunt next considers the various postural hypertonicities: first, the cortical or pyramidal, involving mainly the isolated-synergic movements; which explains why the spastic states are so sharply localized in the distal portions of the extremities; second, the striatal or pallidal types of hypertonus with waxy rigidity, loss of automatic-associated movements and lack of the elements of spasticity, such

as clonus, hyperreflexia, etc.; third, combined pallidopyramidal types of paralysis and spasticity-rigidity; fourth, midbrain or paleocerebellar type of postural hypertonus (decerebrate rigidity as described by Sherrington and Wilson) which involves the severance of the pyramidal tracts (neokinetic) the pallidal system (paleokinetic) and the cortico-ponto-cerebellar mechanism (neostatic), leaving intact the peleo-cerebello-rubro-spinal system (paleostatic). "Decerebrate rigidity may, therefore, be regarded as the postural expression of paleostatic activity." Finally Hunt considers the spinal type of postural hypertonus which expresses itself on the one hand through paraplegia in flexion, due to a transverse cord lesion and involvement of all long tracts, and paraplegia in extension following involvement only of the pyramidal tracts.

WECHSLER, New York.

METHODS TO SECURE END-TO-END SUTURE OF PERIPHERAL NERVES. HOWARD C. NAFFZIGER, Surg., Gynec. & Obst., March, 1921.

Numerous methods of filling in gaps occasioned by nerve division have been recommended by surgeons and, in large part, have resulted in connecting the severed nerve ends by a material not penetrable by the neurons. The experimental works of Huber, Nageotte and others in the use of free nerve grafts have pointed the way to a more rational treatment. With improvement in operative procedures, the necessity for grafts has become considerably decreased, while end-to-end suture has often become more possible.

More than one hundred patients with peripheral nerve injury have come to operation within the past eighteen months from the United States Army Letterman General Hospital, the neurologic service of the University of California Hospital, and from other sources largely industrial. Autonerve grafts, homografts, or heterografts should be reserved for cases in which all methods of securing direct suture have been exhausted. Apparently it is not an uncommon procedure for surgeons to resect bones of the extremities to secure shortening in order that they may obtain end-to-end suture. Conditions justifying this measure must be exceedingly rare.

The most useful methods in obtaining apposition of the divided nerve ends are: (1) free mobilization of the proximal and distal portions of the nerve, (2) transposition of the nerve to a shorter route than the normal one, (3) favorable posture of the extremity to shorten the distance to be overcome, and (4) gradual lengthening of the nerve by a two-stage operation.

Free mobilization of the nerve requires above all long incisions. Small incisions and forcible stretching of the nerve produce unnecessary trauma and does not secure the greatest lengthening.

Transposition of the nerve to a shorter route than the normal can be employed in the ulnar nerve and in the musculospiral, the ulnar nerve being elevated and placed internal to the condyle and the musculospiral placed anterior to the humerus on the brachialis anticus.

Great stress is placed on choosing a favorable posture to give the greatest mobility of nerve eminence in order to permit end-to-end suture, rather than using nerve graft. The author resorts to gradual lengthening of the nerve by a two-stage operation, in extreme cases suturing the severed ends before the neuromas are excised, placing the extremity into a cast or frame, and permitting gradual stretching for a period of two months. End-to-end anastomosis should follow the removal of the neuromas and without rotation of

the nerve axons. The suture line should be surrounded with foreign material, but, if convenient, the suture line should be brought up against uninjured muscle or between muscle planes.

If the arm has been held in flexion or adduction in order to permit an end-to-end anastomosis, it should not be straightened for a period of from five to six weeks and then very gradually in order to permit the extremity to lie in a half cast. Daily massage and gentle passive motion are valuable.

The value of nerve graft is recognized when end-to-end suture cannot be obtained. Such cases, however, are relatively rare and nerve graft should be resorted to only after all attempts at end-to-end suture have been unsuccessful. For the ulnar nerve, mobilization alone permits of suture after a 3 cm. gap; after mobilization and transposition 4.5 cm. Adduction of the arm to the side permits a slight additional gain; with mobilization, transposition, adduction and complete flexion of elbow and wrist, 10 cm. For the musculospiral nerve, mobilization alone permits 2 cm.; with mobilization, elbow flexion and adduction, slightly more, usually about 8.5 cm.; mobilization, transposition, elbow flexion and adduction, 10 cm.

In conclusion, the author states that end-to-end suture is the method of choice and with few exceptions can be employed. This is particularly true of the main nerve trunks. Familiarity with these methods in peripheral nerve surgery gives an increasingly high proportion of end-to-end sutures.

Adson, Rochester, Minn.

STUDIES IN EXHAUSTION: AN EXPERIMENTAL RESEARCH. G. W. Crile, Arch. Surg. **2**:197 (March) 1921.

Dr. Crile had in mind in this research problem, the identification of the organs and tissues affected by various causes of exhaustion, and whether or not all known causes of exhaustion produce identical end-effects in the essential organs.

He states that stimulants are valueless in exhaustion, that restoration time is much the same for all causes and that sleep is the essential for restoration. The primary cause of exhaustion states could not be ascertained.

In the study of insomnia tests were carried out on normal, healthy rabbits. Early and late histologic, biochemical, physical and chemical changes were observed after subjecting the animals to varying degrees of insomnia. The results of natural sleep, artificial, diurnal and seasonal sleep are compared. The animals were kept continuously awake for from ninety-six to one hundred and eighteen hours, without other disturbances and with plenty of food and water at all times.

In the histologic studies the tissues found to be affected were the central nervous system, the liver and suprarenals, all others showing no change. In the nervous system the cortex was more severely affected than other areas, but since the Purkinje cells of the cerebellum showed changes identical with those of the cortex the differential counts were made from these. In the liver the cells near the periphery of the lobules showed most alteration, while in the suprarenals the cells of the cortex alone were affected.

In the differential counts there were recognized active cells, fatigue cells and exhausted cells. It was found that there was early a considerable increase of active cells above the normal (64.8 per cent.). This is termed a period of overreaction or hyperchromatism. The exhausted cells began to increase again after the period of maximum reaction. Animals in one series that were not

killed died soon after the period of insomnia was over. No cause for death could be found and it was concluded that the exhaustion had been carried to a nonsurvivable limit. It is estimated that survival either in man or animal cannot occur after a loss of 15 per cent. of brain cells.

Observations were made during the periods of insomnia and at varying periods afterward. The cell changes consisted mainly of swelling and enlargement of the cells, diminution and disappearance of granules (except in the early hyperchromatic stage), dislocation of nuclei, rupture and disintegration of cells. In regard to the possibilities of restoration, one seance of sleep was found to restore all cells except those disintegrated. In those animals dying from the insomnia, 15 per cent. of the brain cells were fully exhausted. In the cases in which artificial sleep was produced (nitrous oxid-oxygen anesthesia) 90 per cent. of the cells became hyperchromatic. From the latter it was concluded that sleep repairs all cells except those in which the nucleus has disintegrated, and that artificial sleep is markedly beneficial.

From the physiochemical point of view hydrogen-ion concentration determinations were made, it being known that intravenous injections of sodium phosphate or hydrochloric acid produced lesions in the brain, liver and suprarenals identical with those produced by exhaustion. The hydrogen-ion concentration was determined in six rabbits before and after insomnia. The conclusions arrived at were that prolonged insomnia alone produces relatively slight or no alteration.

Electrical conductivity experiments led to the conclusion that prolonged insomnia (ninety-six hours) decreased the conductivity of the central nervous system. After a period of rest (six hours), the conductivity of the cerebrum and cerebellum were decreased still more than at the close of the insomnia period, but the spinal cord had begun to return toward the normal. After a prolonged rest (from nine days to two weeks) the cerebral conductivity was increased above the normal, while that of the cerebellum and spinal cord approached normal.

The body temperature in exhaustion in 42 cases was found to rise an average of 1.5 F. in 24 cases; to fall 0.9 F. in 14 cases; while in 6 there was no change. The respiratory rate showed an average increase of 47 per minute in 6 cases, a decrease of 46 in 31 cases and no alteration in 5 cases. The weight in 23 cases showed an average gain of 107 gm.; a loss of 107 gm. in 18 cases, and no change in 1 case. Urinalysis in 17 cases was negative for pathologic constituents.

The article is illustrated by photomicrographs of the cellular changes, and there are tables of each experiment which are worthy of careful study. In the light of the results obtained an interesting speculation arises concerning the condition of the essential organs in those cases of maniacal excitement in the manic-depressive psychoses, in which patients go for days and even weeks without sleep or rest and are subjected to the most extreme grades of emotional imbalance (which the author was careful to avoid in his experiments), which apparently so completely controls their activities. Neuropathologists are accustomed to the pictures of marked cellular changes in toxic states and terminal conditions. Can death be due to the toxicity of the disease or does exhaustion beyond a survivable limit bring about the fatal termination? In the treatment of all acute disturbances, physical as well as mental, are not sleep and rest, however produced, the greatest therapeutic agents at our command? The author's experiment has certainly opened up a field of inquiry which is rife with possibilities.

PATTEN, Philadelphia.

A CASE OF PSEUDOTETANUS (ESCHERICH). T. Reh, Rev. neurol. **27**:1017 (Oct.) 1920.

In presenting his case, the author remarks on the apparent clinical infrequency of this condition, only twenty cases having been described thus far in the literature.

Reh's patient, a girl of 3, was brought to his clinic three days after the onset of a series of severe clonic seizures, at first occurring every fifteen or twenty minutes, but by the time of presentation having become somewhat less severe and definitely less frequent. The condition was reported to have been ushered in by cervical rigidity, which gradually became generalized. The family and personal history was entirely negative with the exception of a report of a small cutaneous laceration (lesion still in evidence) received some two weeks previously as a result of a scratch from a ground bramble.

Examination showed in addition to occasional and rather mild clonic seizures, a marked generalized, plank-like rigidity affecting the entire body except that the ocular muscles were practically uninvolved and those of the arms only slightly so. This condition was apparently unaccompanied by pain or any indication of acute subjective distress. The eyelids were noted to be half closed and the eyebrows drawn, with marked wrinkling and creasing of the forehead and cheeks. Trismus was definitely present. Physical and neurologic examinations were otherwise negative, and there was no indication at this time, or at any other during the later course of the illness, of the Weiss, Chvostek or Trousseau signs or evidence of changed electrical reaction, or report of fever. The spinal fluid was negative, and it was impossible to isolate the tetanus bacillus from the skin lesion. Shortly after examination 20 c.c. of tetanus antitoxin were administered.

The clinical course was one of gradual and uneventful improvement, the patient having completely recovered by the twenty-eighth day after admission to Reh's service. Nosologically the author concludes that, although the clinical duality of tetanus and pseudotetanus seems fairly well established, the etiologic status of the latter is still definitely obscure, and he recommends that this condition be regarded rather as a clinical syndrome dependent possibly on a number of etiologic factors than as a truly specific disease entity.

Raphael, Ann Arbor, Mich.

SYPHILITIC BACKACHE: A SYMPTOM OF SYPHILIS OF THE SPINAL CORD, LUMBAR MUSCLES AND VERTEBRAE, WITH REPORT OF CASES. J. V. Klauder, Arch. Dermat. & Syph. **3**:761 (June) 1921.

Klauder discusses backache from the standpoint of neurosyphilis, syphilis of the muscles, vertebrae and synovial involvement of the joints. Most of the syphilitic backaches arise from syphilitic involvement of the spinal cord. A meningeal inflammation causes irritation of the posterior roots or the inflammation may extend to the root itself causing pain. Vascular disease, localized gummatous processes or syphilitic periostitis by extension may cause pain. Meningeal irritation and inflammation is more extensive and intense in the posterior columns and surfaces of the cord, the dorsal region having least resistance suffering most. Meningeal inflammation may exist from the exanthematous period of syphilis and last many years. Headaches and backaches of secondary syphilis are frequently meningeal in origin. The meningeal syn-

drome is: pain, paresthesias, hyperesthesia and increased tendon reflexes. The patellar tendon reflexes are often variable, due probably to the swelling around the roots from the acute syphilitic process. The early opinion that syphilitic pain was worse at night was not borne out in the author's experience. The patients themselves complain chiefly of backache, fatigability and stiffness. Objectively there are found spinal rigidity, localized or diffuse tenderness on percussion over the spinous processes and painful movement. Extension of the syphilitic process causes the familiar picture of meningomyelitis. Antisyphilitic therapy produces a response proportional to the duration and the stage of the disease, and spinal fluid examinations are frequently positive when the blood Wassermann reaction is negative.

Lumbar muscle involvement occurs in the secondary stage of syphilis, either meningeal in origin or as a spirochetal toxemia with fever, headache and malaise (simulating typhoid). The pathologic changes are: interstitial myositis —attended by swelling and pain, gummatous formations—localized or generalized and accompanied by pain, infiltration and swelling and perhaps ulcer formation.

Vertebral involvement is rare in syphilis, but when it occurs it is usually in the cervical region. It is seen as a periostitis or osteitis. The predominant symptom is pain—acute and lancinating; there are also tenderness, rigidity and obliteration of the normal curve. Deformities occur. Differentiation from tuberculosis is the main concern in diagnosis. Frequently, however, syphilitic infection serves to light up a quiescent tuberculous process.

Syphilitic synovitis is the commonest lesion of spinal syphilis. It occurs early in the disease, affects three or four vertebrae and has for symptomatology: deformity, localized stiffness and hypotonicity of the uninvolved parts. Roentgen-ray evidence is usually wanting.

Case reports are given illustrative of each type of syphilitic involvement and photographs accompany the text.

PATTEN, Philadelphia.

THE FINGER-FLEXION SIGN IN HEMIPLEGIA. T. URLATZEANU and D. NOICA, Rev. neurol. **27**:1012 (Oct.) 1920.

The authors direct attention to a feature especially noted by them in recent hemiplegias, dependent on cerebral embolus, thrombosis or hemorrhage, which they believe to be of definite diagnostic significance. Thus it will often be observed, in such patients, that when in the supine position, with the hand of the affected side resting on the bed in slight pronation, it will be seen to be supported on its cubital border with the thumb approximated to the index finger with consequent prominence of the thenar eminence and wrinkling of the skin over the palmothenar area. The terminal phalanx of the thumb is gently flexed in contradistinction to the normal position, that of extension. The other fingers, particularly the last two, are approximated to one another and are held gently flexed. The forearm at the same time is generally found to rest in a position of semipronation. Later in the condition, if recovery does not take place, permanent contracture in this position frequently occurs. This flexion attitude tends to reinstate itself automatically if altered by the examiner. It has been observed that, if the arm is set at an angle of 90 degrees from the trunk, and allowed to hang over the side of the bed, although the forearm and hand are now in supination, the fingers still retain their former flexion position. Urlatzeanu and Noica have been unable to determine any analogous condition as occurring in normal persons, although a

similar state, but bilateral, has been noted by them in patients in a state of anesthesia or in coma, either toxic or infectious. The exact mechanism underlying this posture sign is somewhat obscure, although the authors suggest relation, in this connection, to lesion in the opposite cerebral hemisphere.

RAPHAEL, Ann Arbor, Mich.

AN EXTENSIVE SPINAL ARACHNOID FIBROBLASTOMA. HUBERT S. HOWE, Neurol. Bull. **3**: No. 6 (June) 1921.

Howe reports an unusual type of cord tumor which involved practically the whole cord from the filum terminale up to the lower cervical segments. The condition lasted twelve years from the onset of the first symptom to the date of the patient's death. The first symptoms were those of a tumor of the cord, which though apparently incompletely removed (seventh cervical to the fourth dorsal) resulted in complete clinical recovery for nearly five years. A second operation, eight years after the first, resulted in fair recovery, and a third, three years later, ended in the patient's death. At necropsy the cord was found "imbedded in an irregular tumor mass which was most abundantly present in the sacral region and about the filum terminale, but extended upward as high as the upper dorsal region." Tumor tissue was found also near the fourth cervical segment, although it gave no signs of compression. The unusual features of the case were the immense dimensions of the tumor and almost complete recovery following incomplete removal on two occasions. Another unusual feature, which is difficult to correlate with the involvement of the cord, was the presence of choked disk in the absence of signs of brain involvement and negative necropsy findings on gross examination. An undetected metastatic focus in the brain may possibly be conjectured as the cause of the choked disk, but the findings recorded hardly warrant such a conclusion.

WECHSLER, New York.

HISTOLOGIC EXAMINATION OF THE NEURAXIS IN A CASE OF SYDENHAM'S CHOREA. P. MARIE and C. TRETIAKOFF, Rev. Neurol. **27**:428 (May) 1920.

Marie and Tretiakoff present the case of a girl, aged 10, who died ten days after the onset of what appeared clinically as typically Sydenham's chorea, the pathologic findings in which agreed very well, on the whole, with those determined in similar cases previously reported.

Histopathologic study revealed a somewhat diffuse and irregularly distributed inflammatory reaction, primarily perivascular, but also showing definite parenchymatous invasion (particularly the gray matter) and affecting the entire neuraxis (except the bulb and cerebellum), focusing maximally, however, on the basal ganglions—in short, a picture essentially that of a polioencephalitis. As such, this reaction seems to have been essentially irritative, marked by intense hyperemia and leukocytic (mononuclear) infiltration, in association with neuroglial and fibroblastic proliferation. Degenerative change seems to have been definitely of secondary prominence, by which, and the severity of the medullary involvement, differentiation may be made pathologically from acute anterior poliomyelitis as well as Landry's paralysis, rabies and tetanus. Differentiation from epidemic encephalitis, however, is extremely difficult, although, as a rule, in the latter the cortical change is less marked and the oculomotor nuclei are rarely uninvolved.

The authors direct attention finally to the similarity between the findings in their case and those determined by Marie and Lhermitte in Huntington's chorea particularly in point of involvement emphasis upon the cerebral cortex and corpus striatum, and maximally upon the latter. This, being an outstanding feature in both affections, throws into signal relief the possible significance of striate disorder in the genesis of choreiform movements so-called.

RAPHAEL, Ann Arbor, Mich.

EPIDEMIC HICCOUGH. A. HANNS, Progrès med. **36**:257 (May 28) 1921.

The nature of epidemic hiccup is still obscure but tends, more and more, to be considered as an elective and solitary localization of attenuated virus of epidemic encephalitis.

The chief arguments raised against the identity in the nature of these two affections have been that the hiccup always, it is claimed, exists alone, and that inversely in a case of epidemic encephalitis among its so numerous and varied manifestations, the hiccup has not been observed. Such arguments are refuted, however, by the case which the writer takes occasion to report.

In this case there was persistent hiccup, more or less constant, from the seventh to the twenty-third day of an afebrile illness in which headache, photophobia, somnolence (lasting twenty-one days), paresis of an arm, facial paralysis, ptosis and dysarthria were concomitant symptoms. The hiccup involved diaphragmatic contractions, as well as symmetrically bilateral contractions of the scaleni muscles.

The diagnosis was accepted as encephalitis. The case appeared to prove that hiccup can be a part of the symptomatology of encephalitis and tends to show that monosymtomatic hiccup is a "forme fruste" of an encephalitis.

DAVIS, New York.

CYSTICERCUS SPINAL MENINGITIS. TITUS VASILIU, Presse méd. **29**: 522 (July 2) 1921.

The case described was first diagnosed as spinal cord tumor, between the seventh cervical and second dorsal segments. Necropsy revealed an enlargement of the whole cervical cord due to a grossly thickened pia, from 2 to 10 mm. in thickness. The microscopic appearance suggested tuberculosis. Microscopically the vessel walls were found markedly thickened; the intima was rich in connective tissue cells, leukocytes and giant cells ("giant-cell endarteritis"); the lumina contained plicated cysticerci. No diagnosis could be made between a *taenia solium* and a *saginata;* hooklets were not found. No worms were present in the intestine.

Askanazy (*Deutsch med. Wchnschr.*, 1902, p. 431) is quoted as having found cysticerci only outside the vessels, and Jacobsohn (*Monatschr. f. Psychiat. u. Neurol.* **21**:119) as finding the walls of cysticercus capsules made up of the walls of small vessels. Vasiliu believes that the particular histologic picture in his case is unique; besides the presence of such an endarteritis, the limitation of lesions to the meninges without invasion of the nerve tissue is noteworthy.

HUDDLESON, New York.

CONCERNING THE INTRASPINOUS TREATMENT OF NEUROSYPHILIS. SICARD and PARAF, Rev. neurol. **27**:1033 (Oct.) 1920.

Comment is made on the favorable results reported by other workers, notably Marinesco, Swift and Ellis, as attained by the treatment of neurosyphilis with autoarsphenamized serum, intraspinally administered. This seems to be somewhat contrary to the authors' experience, since they noted in a series of fourteen cases carefully observed over a long period (mainly tabetic and paretic patients) that practically no improvement followed such treatment except in cases in which the subdural schedule was combined with general intensive intravenous and intramuscular therapy and that, in addition, in this series, there occurred two sudden deaths shortly following subdural injections. The authors feel the chief value of the intraspinal technic to lie in the induction of increased meningeal permeability, thus allowing for the freer entry into the neuraxial field of medication reaching it through the blood stream and suggest as safer and more efficient in the treatment of neurosyphilis, the intraspinal administration of simple sterile saline (0.7 per cent. in amounts of 4-10 c.c.) in intensive association with general intravenous and intramuscular therapy.

RAPHAEL, Ann Arbor, Mich.

EPIDEMIC ENCEPHALITIS AND PREGNANCY. MARINESCO, Bull. de l'Acad. de méd. **85**:34 (July) 1921.

Two cases of pregnancy, complicated by epidemic encephalitis, show that pregnancy and delivery appear to render the encephalitis more serious.

The apparition of epidemic encephalitis in the course of pregnancy exercises an influence on it comparable to that of most infectious diseases. In some instances, the pregnancy can complete its course and the new-born remain free from any contamination. In other instances, the encephalitis exercises an unfavorable influence and brings about an interruption of the pregnancy; an interruption probably due to a direct intervention of the virus by way of the placenta.

In two cases (one reported by Harris and one by Novals and Sousa) of encephalitic symptoms in new-born children, the disease was very light, for it was over at the end of a few days. This argues an attenuated virus.

This attenuation of placental transmitted virus makes it seem possible that when the death or expulsion of the fetus occurs, factors other than placental contamination, such as prolonged hyperthermia and disturbances of metabolism metabolism are also at work.

DAVIS, New York.

THE SACHS-GEORGI REACTION FOR SYPHILIS. A PRELIMINARY REPORT OF MORE THAN ONE THOUSAND COMPARATIVE WASSERMANN AND SACHS-GEORGI TESTS. S. A. LEVINSON and W. F. PETERSEN, Arch. Dermat. & Syph. **3**:286 (March) 1921.

The paper gives the technic of this test and discusses it in comparison with the Wassermann. For the technical part the reader is referred to the original. The authors state that in an examination of 1,042 serums and cerebrospinal fluids by both the Wassermann and the Sachs-Georgi reaction there was an agreement in 92 per cent. In sixty-two cases, 58 per cent. of which gave positive clinical evidence of syphilis and in which the Wassermann reaction

was negative, a positive or doubtful reaction was obtained by the Sachs-Georgi method. The conclusions reached are that the technic of the Sachs-Georgi is more simple than the Wassermann test, more uniform results are obtained and in routine work it is of invaluable aid when done in conjunction with the Wassermann test.

PATTEN, Philadelphia.

HEAD TRAUMA. H. BILLET, Progrès med. **36**: 323 (July 9) 1921.

The writer believes that there should be systematic surgical exploration of every scalp wound and trepanning if the exploration shows even slight solution of continuity of the cranial wall.

In states of trauma without scalp injury, most careful examination of the cranial vault for depression and examination of the patient for any signs of localization are necessary, as well as lumbar puncture in all instances. Do not fear taking an appreciable quantity of spinal fluid, even from 30 to 50 c.c. Stop only when the liquid no longer flows with pressure.

If the removal of the spinal fluid aggravates the symptoms, it indicates that a double subtemporal trepanning is advisable. If the condition rests the same or improves under lumbar puncture, it is advisable to hold to these, repeating them either once or twice daily, until the condition is ameliorated.

DAVIS, New York.

A FOCUS OF CEREBRAL SOFTENING IN THE COURSE OF ACUTE TUBERCULOUS MENINGITIS. L. LORTAT JACOB and R. TURPIN, Progrès méd. **36**:357 (July 30) 1921.

The authors detail these clinical facts: In a man aged 57 with pulmonary tuberculosis, there developed neck rigidity, Brudzinski and Kernig signs, fever, depression and subsequently somnolence. The spinal fluid conformed to that of tuberculous meningitis. In the third week of these meningeal symptoms, twenty-four hours before death, pyramidal tract signs appeared on the right side.

Necropsy showed that apart from the manifest lesions of the tuberculous meningitis, there was an area of subcortical softening of the left cerebral hemisphere. The writers review briefly the subject of tuberculous involvement of encephalic vessels and conclude that in their case the focal lesion found was due to a local tuberculous endarteritis.

DAVIS, New York.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Eighty-Ninth Regular Meeting, May 3, 1921

FOSTER KENNEDY, M.D., *President, in the Chair*

RESOLUTIONS ON THE DEATH OF DR. HYMAN CLIMENKO
1873-1921

Hyman Climenko was born in Russia. He received his preliminary education in that country, having attended the "Gymnasium," a secondary school, up to the time he emigrated to America in 1890. Economic necessity forced him to discontinue his studies for a time, but subsequently he resumed them and entered Long Island Medical College in 1900, being graduated from that institution in 1904.

After a period at the Montefiore Home he went into general practice, but kept up his neurologic studies by associating himself with Dr. Joseph Collins at the New York Post-Graduate Medical School and Hospital, and later at the Neurological Institute. About ten years ago he was appointed adjunct visiting neurologist to the Montefiore Home, later chief of clinic at the Mt. Sinai dispensary, and shortly before his death adjunct neurologist to the Mt. Sinai Hospital and full visiting neurologist to the Montefiore Home and Hospital for Chronic Diseases. He was a member of numerous medical societies, among them the New York Neurological Society and Academy of Medicine, and two days before his death was elected chairman of the Section of Neurology and Psychiatry of the Academy.

Dr. Climenko's death came as a shock to his friends. In perfect health, he was taken ill with acute abdominal pain. The condition was diagnosticated as acute intestinal obstruction; he was immediately operated on but he failed to rally and died within three days from a severe toxemia. He is survived by his widow and five children.

Hyman Climenko was an able physician and an excellent neurologist. He was rapidly rising when his untimely death put an end to the fond hopes of his friends and his own aspirations. Since 1908 he had contributed forty papers on neurological subjects. A short list of titles includes Anterior Poliomyelitis, Familial Optic Neuritis, Tumors of the Brain and Cord, Dystonia Musculorum, Infantilism, Syringomyelia and Leprous Neuritis, Corpus Luteum in Neurological Practice, and Lethargic Encephalitis. His work combined scholarship with accuracy of observation.

Hyman Climenko was a most lovable man. He had a big heart, was a true and devoted friend, literally had no enemies, and was ever kind and considerate. He was so modest that he never made a strong first impression, but close acquaintance quickly revealed the sterling man. Beside his medical activities he showed great interest in liberal social and literary movements. In him the New York Neurological Society has lost a valuable member and an unusually diligent and industrious worker.

I. ABRAHAMSON,
I. STRAUSS,
I. S. WECHSLER,
Committee.

DR. FOSTER KENNEDY wished to say he had enjoyed knowing Dr. Climenko. He was a gallant fellow, a happy fighter against large odds and an intellectually honest man. Climenko possessed also a quaint and whimsical humor, and enjoyed nothing better than to be lightly chaffed himself. He was a very lovable and a very real person, and Dr. Kennedy felt that the Society had suffered a great loss in his untimely death.

A PSYCHOGENIC STUDY OF EPILEPSY IN A CHILD. DR. L. PIERCE CLARK.

Dr. Clark said that since epileptoid seizures, observed in psychoneurotic patients as early as the time of Westphal, have been favorably influenced by psychotherapy, the same method has been tried on essential epilepsy. Essential epilepsy, however, is not a neurosis but is organic in origin, and the results have not been good. Nevertheless, cases appear in which psychogenic factors so clearly overlie other causative agents that the question arises whether these trends may not be of prime etiologic and therapeutic importance.

The personality makeup of the epileptic child removes him so far from natural development that ordinary adjustments are impossible. Coincident with grand mal seizures many epileptic patients show psychopathic episodes. While these are related to their defective personalities as a whole, they are not directly related to the seizure episodes themselves, yet when these are immediate forerunners or may in themselves constitute imbricated attacks or petit mal, they are of greatest moment.

The case described is that of a boy, now 5 years old, who at the age of 2½, eight months after the birth of his younger brother had attacks which were for the most part conscious. He began to clench his fist and grimace, ending with laughing and crying. A year later the symptoms became more severe until the attacks were not only unconscious but ended in classic grand mal. A very active child, nervous and irritable from birth, of neurotic and high strung parentage, he from the first was jealous of his brother. Two types of attacks were noted, one the ordinary grand mal. About two thirds of his severe seizures were preceded by laughing or puffing attacks. The boy suddenly stood quite still and breathed or puffed as one might on entering a cold bath. The lips were drawn back and upward giving a leering expression. The smiling contraction of the lower part of the face had the suggestion of animal snarl. The eyes had a vacant, fixed, far-away gaze; the pupils dilated and were unresponsive to light for the eight or ten seconds of the attack. On one occasion when put to bed after several puffing attacks he had two minor attacks in rapid succession following displeasure. When he wanted to get up and was restrained, he threatened to have another. Instead of the usual puffing attack he merely leered and threw his arms and legs about in utter abandon. The following night while asleep he began drawing in his breath in stertorous gasps; his limbs were in tonic spasm, then clonic, and finally movement ceased as in a fairly severe grand mal. The latter would seem to have started as an ordinary puffing attack and gradually shaded over into a moderately severe grand mal. He did not waken after the attack until morning when he felt quite well. Two nights later he had a complete grand mal attack which also began as a respiratory or leering episode. A few weeks later, when annoyed, he simulated an attack, but it was a poor imitation and he admitted the hoax. Tantrums and an increasing number of nocturnal attacks, both puffing and grand mal, together with wishing spells of very childish nature, followed.

Since he was showing increased fatigue, irritability, and more attacks both mild and severe, it was concluded that his effort at attention in school was too intense and he was placed on a less taxing routine. Improvement was at once apparent, but whenever any unpleasant task was imposed he would start puffing and blowing. He admitted that he encouraged the attack by these actions, that this as well as the peculiar attack itself made his "stomach light" as though he were "floating." He would not say the acts were pleasurable, but said they gave him relief. After a time his school work was good, and he did well in his daily routine. He said he did no more puffing now "because things were going all right and he didn't need to." Whenever the routine became irksome the tantrums or attacks or wish episodes, would occur. Apparently the conscious puffings were employed to while away the general tedium of life or displeasurable states that were to be abreacted. The act was usually conscious at its initiation and if persisted in became an unconscious laughing crying attack and, if this was not controlled or aborted, after two or three of them had occurred the whole episode might end in a classic grand mal.

Remarkable improvement has been noted in the past four months. Tantrum episodes rarely appear, and evidences of a desire to retreat from reality in his wish fantasies and in puffings are eliminated; the grand mal has been absent now for over four months. The retreat, or displeasure mechanism, was apparently first employed at the birth of his brother when his mother's attention was diverted from him. The boy shows the grand mal epileptic in the making, and that the disorder is based on a defective epileptic anlage of constitutional origin. The personality uses the defective instinct of adaptation for its own ends—a conscious and unconscious retreat from reality, which is proportionate to the stress demand.

DISCUSSION

Dr. Smith Ely Jelliffe said that he agreed absolutely with Dr. Clark. Observation from day to day, training and morale adjustments, all are important, and we should be in a better position to treat the kinds of attacks that are usually lumped together under the title of epilepsy.

Dr. Clark said that the personality of the little boy had undergone a remarkable change in the course of his observation. His physiognomy and voice, manner of walking, have all changed. It was thought previously, by his family, that he was not so different from other children, but with his improvement they are surprised that they did not see his shortcomings before. The difficulty now encountered is that of injecting the boy into his home environment when his course of treatment is completed. Transplantation into a different family may be necessary for a number of years. Even though the relatives might be trained and think that they understand the situation, they would nevertheless react to family type.

EPIDEMIC ENCEPHALITIS SIMULATING BRAIN TUMOR. Dr. Irving J. Sands.

These cases from the Psychopathic Service of Bellevue presented all the general signs of brain tumor, such as headache, choked disks, hemiplegia and vomiting of the projectile type. In two of the cases operation had been decided on but permission for operative interference was refused, in one instance by the patient herself and in the other by the patient's parents. The third case terminated fatally.

Case 1, a boy of 12, of negative family and personal history, in June, 1920, began to show loss of interest in his surroundings, with awkwardness in the use of the left hand. Then followed a steadily increasing paralysis of the left side of the body which later spread to the right side, with choked disks, first in the right and later in the left eye. The paralysis was at first flaccid and later spastic. He lost control of his sphincters and had dysphagia at first for solid and later for liquid food. The mental picture was that of indifference, but the patient responded to questions, and his answers were relevant and coherent and his emotional reactions adequate and consistent. Serologic examinations excluded syphilis. Permission for operation was refused by the parents. The patient began to recover in the middle of September, 1920, and was discharged from the hospital in the middle of November, 1920. He was last seen in March, 1921, had returned to school and was getting along fairly well except that he was quite readily excited and irritated and would fly into rages. Physically he showed the residuals of an old left hemiplegia, with slight haziness of the right disk, and there were definite involuntary choreo-athetoid movements in the fingers and hand of the left side on voluntary motion of any other part of the body.

Case 2 was that of a 42-year old married woman of negative family and personal histories. In the middle of February, 1921, she began to complain of general malaise, headache and a slight cough. On March 1, she suddenly took to bed, refused to talk and would see no one. In the hospital she showed evidence at first of right hemiplegia, with choking of the left disk. Later the process extended to the right side of the brain producing greater choking in the right than in the left disk, with hemorrhage in both disks. Subsequently she showed considerable meningeal irritation. Serology was negative for syphilis. A roentgenogram of the skull was negative. The mental picture was that of steadily increasing drowsiness, lack of attention and inability to answer questions. She lost control of the sphincters and regurgitated even liquid food. She died on April 14. Necropsy was refused.

In Case 3, the patient from the very beginning showed evidence of a growth in the right side of the brain, causing left hemiplegia with elevation of the right disk. Serology excluded syphilis. She had periodic attacks of headache, nausea and projectile vomiting. Her mental picture was characterized by marked irritability and confusion. The physical signs pointed so strongly toward a cerebral neoplasm that epidemic encephalitis was not even considered as a possibility, and she was transferred from the psychopathic service for operative interference. She refused permission and began to improve. After ten weeks she showed practically no residuals of her illness.

Dr. Sands referred to the works of Bramwell, Barker, Tilney and Howe, as evidence that changes in the disks in cases of epidemic encephalitis were not usually encountered, and choked disks were indeed rare findings in this disease. He explained the choked disk on the theory that there was pressure on the supra-optic canal of Tilney, rather than by the usual theories—such as the toxic theory of Leber or the mechanical theory of Schmidt-Rimpler.

DISCUSSION

Dr. Smith Ely Jelliffe recalled a case which he had considered one of brain tumor until Dr. Kennedy pointed out to him that it was epidemic encephalitis. In still another case the picture was one of fractured base of the skull. The patient had fallen on the first day of the illness, blood was found on cerebrospinal puncture and the physicians were sure of fracture. The illness pursued a regular encephalitis course and gradually cleared up.

Dr. I. Abrahamson thought that the course of the disease was essentially different in encephalitis and in cerebral neoplasm. Even the papillitides in these conditions had different onsets and different rates of progress. Left hemiplegia in a right-handed person with aphasia indicated multiplicity of the lesions.

In encephalitis, mental signs were common, quite marked and occurred early in the disease. In brain tumors they were less evident and usually were found late in the disease or when much internal hydrocephalus existed, the exceptions being chiefly temperosphenoidal neoplasms. He cited the case of a girl aged 12 years, with optic neuritis, paraplegia—at first left and later right sided; at first flaccid, then spastic, then flaccidity plus tremors, coincident with return of power in the extremities. There was bulbar involvement; the Wassermann reaction of the blood was ++++; the spinal fluid was negative. Epidemic encephalitis was finally diagnosed.

Another case was that of a young woman with markedly choked disks from 4 to 5 diopters, hemiparesis, ocular signs, etc. She gave a history of an infected pigmented mole on the left leg which had been removed; then a gland had been removed from the left groin, diagnosed as a melanocarcinoma. The differential diagnosis of metastatic carcinoma of the brain and an epidemic encephalitis was decided in favor of the latter; this was confirmed by necropsy.

The importance of the mental states of patients with epidemic encephalitis had to be constantly borne in mind in making a differential diagnosis.

Dr. Foster Kennedy remarked on the interesting feature of the papilledema, its suddenness and lateness in the course of the disease. It appeared over night. The maximum was reached in twenty-four hours. Such speed of progress is not seen in any other condition. The vision was obscured with scotomas. It had not been easy to discriminate between tumor and encephalitis.

The type of encephalitis described by Dr. Sands is new, though it is not appearing frequently. It is often associated with infective states. It seems to be a phenomenon of these times, and is possibly a condition following the influenza epidemic.

Dr. Sands, in closing, said that while the clinical pictures of epidemic encephalitis that have lately been reported are quite different from those reported two years ago, one would not hesitate to declare them to be caused by the same organism. The mental picture is a great aid in arriving at the correct diagnosis. The apparent symptoms must be differentiated from the real ones. While these patients seem to be in a state of lethargy, on closer observation they are often quite keen and responsive to questions. In brain tumor cases, on reaching such an apparent or real state of lethargy, the patient would never be able to respond to questions and to cooperate for an examination as readily as in cases of epidemic encephalitis simulating cerebral neoplasms.

THE SIGNIFICANCE OF THE REPORTED NERVE CELL ALTERATIONS IN DEMENTIA PRAECOX (WITH LANTERN SLIDES).

Dr. Clarence O. Cheney.

The speaker called attention to the nerve cell and other cortical changes in cases of dementia praecox, as described by various observers, particularly Mott, who has maintained that the nerve cell alterations were responsible for the clinical symptoms of this mental disorder. With the desire to determine the validity of such claims, the speaker had undertaken histologic cortex studies

of a series of dementia praecox cases, with another series of manic-depressive cases for control, using photomicrographs extensively. The comparative cortical appearances in the dementia praecox and manic-depressive cases were demonstrated by lantern slides. Conclusions were reached to the effect that in cases of long-standing dementia praecox a nerve cell loss was not evident as compared with cases of manic-depressive psychosis of similar age and cause of death; that frequently identical nerve cell changes were found in manic-depressive cases and cases of dementia praecox; that a marked variability in the nerve cell appearances was present not only from case to case but in different microscopic fields of the same case; that there was no uniform or constant cortical or individual nerve cell picture in dementia praecox which would enable one to distinguish such a case from one of manic-depressive psychosis. It was considered as unproved that the nerve cell changes found were essential to the clinical picture of dementia praecox as it could not be definitely stated how much of these changes might be due to antemortem physical disease, postmortem alterations or technical procedure. It was stated that the study had emphasized the importance and necessity of obtaining dementia praecox brain material which had not been affected as far as could be known by acute or chronic physical disease, and which was as free as possible from the objection of postmortem change, and of comparing this with similar material from normal persons. It was felt that until positive results should be found in such dementia praecox material as compared with normal material we would hardly be justified in looking on dementia praecox as an organic brain disease.

Book Reviews

THE MANNER OF MAN THAT KILLS; SPENCER-CZOLGOSZ-RICHESON. By L. Vernon Briggs, M.D., Director of the Massachusetts Society for Mental Hygiene, Member of the American Institute of Criminal Law and Criminology, Société Medico-Psychologique of Paris, France; New England Society of Psychiatry, American Medico-Psychological Association. $5.00 net. Pp. 440. Boston: Richard G. Badger, Publisher, The Gorham Press.

This is a study of the life history of three famous criminals. They are famous because their crimes were picturesque, attracting a great deal of local attention in the case of two of them and in another national notoriety because the victim was the President of the United States. It is these adventitious circumstances that give special interest to these histories; in their essence and in the genesis of the crime they do not differ from the ordinary variety of criminal records.

The book is published primarily for the purpose of emphasizing the mental and nervous defects in these murderers that the public may be stimulated to renewed efforts to search out the defective and so far as possible segregate and treat him. This is the aim of mental hygiene everywhere, but unfortunately it is a task so difficult and of such enormous proportions that little headway can be made. This book will be of most value to the jurist and to lay persons who need to study the life histories of such defectives to get the proper point of view. It will be of less value to the psychiatrist who meets these cases in his daily work.

The most picturesque of the three cases is that of Bertram D. Spencer, who was executed Sept. 17, 1910, for the murder of Miss Blackstone. It would be difficult to find in criminal annals a more striking career than that of Spencer. The family history is replete with insanity and nervous diseases of one sort and another. His father was a man of ungovernable temper and frequently punished Spencer in a cruel and inhuman fashion. As a boy he was subject to attacks of ungovernable anger. These attacks persisted into adult life and were brought on by trivial situations. For the most part he was affable and was kind to his wife and child. Approximately two years before the murder a number of burglaries occurred in Springfield, Mass. Fourteen of these crimes were definitely traced to Spencer. They were all committed in about the same way. He would enter a house, not necessarily when the inmates were asleep, but walk boldly in and insist on their giving him some money and then would ransack the house for valuables. The lower part of his face was always concealed by a dark handkerchief. He carried a revolver and threatened the inmates. He seemed to prefer being seen while he took enough money and some valuable articles. He would frequently pass by things of great value to pick up objects of little or no worth, such as collar buttons and small articles of jewelry. After he was apprehended more than one hundred such articles were found in his home. His last burglary resulted in the killing of Miss Blackstone, for which he was finally apprehended, tried and subsequently executed.

A difference of opinion as to how Spencer should be classified would be pardonable. Some might call him a psychopathic personality. He certainly was of subnormal intelligence, but how low he was in the intellectual scale

we do not know, as there were no formal tests made of his intellectual stature. Perhaps that midwestern diagnosis of "feeblemindedness with a touch of praecox" would come as near a description of Spencer as any other term.

At his trial abundant evidence was introduced showing his defective heredity, want of self-control, and competent witnesses testified that he was irresponsible. This resulted in his being sent to the Bridgewater Asylum for observation. The monthly reports that were sent to the justice who committed him for observation described fully his defects with repeated opinions that he was insane. After having been under observation for more than a year, the superintendent seems to have discovered that there was a difference between medical and legal insanity, so that he then certified that he was "legally sane." Accordingly he was remanded for trial.

The least interesting of the cases is that of Czolgosz. There is a full description of the methods which were employed to facilitate Czolgosz' journey to the electric chair. There is little question that Czolgosz was suffering from dementia praecox. The disease began about three years before he killed President McKinley. This was rumored at the time, though it was generally claimed that Czolgosz was an anarchist. There is little evidence that he ever was an anarchist, and both he and his crime were disowned by that cult.

From a psychiatric standpoint the case of Richeson has the greater interest. It would be difficult to make a diagnosis of his defect in the absence of a thorough psychic analysis. Richeson was a clergyman with a rather bizarre history, but brilliant and affable. He was a man with a good education, but he had strong sexual impulses. He procured the death of a woman to whom he was engaged by administering to her or persuading her to take cyanid of potassium. All his life he had been subject to severe attacks which were variously described as hysterical, in which he would talk irrationally and incoherently and which were accompanied by amnesia.

At first he denied his guilt, and when arraigned he pleaded "not guilty." Subsequently in a letter to the prosecuting attorney he acknowledged his guilt and changed his plea to "guilty." He was sentenced to be electrocuted, and this without a trial before a judge or jury. While confined in the jail, he succeeded in castrating himself with a tin cover of a preserve jar from which he nearly died of hemorrhage.

THE CLINICAL EXAMINATION OF THE NERVOUS SYSTEM. By G. H. MONRAD-KROHN, M.D. (Christiania), M.R.C.P. (Lond.), M.R.C.S. (Eng.); Lecturer in Neurology at the Royal Frederick University, Christiania, Physician to the Neurological Section of the State Hospital, Christiania, also in Charge of the Hospital's Out-Patient Department for Nervous and Mental Diseases; with a Foreword by T. GRAINGER STEWART, M.D., F.R.C.P., Physician to Out-Patients, National Hospital for the Paralyzed and Epileptic, Queen Square; Physician to the West London Hospital; Physician to the Central London Ophthalmic Hospital; Neurologist to the Special Surgical Hospital (Ministry of Pensions). Price, 6 shillings. Pp. 129. H. K. Lewis & Co., Ltd., 1921.

This opusculum of only about 3,500 words (excluding foreword and preface) contains an astonishing amount of information. The author, a Norwegian writing English, evidently has turned his back on the polysyllabic cult. His point is clearly stated in a few short words. Verily it is restful to read English which is correct, yet aspires to be neither eloquent, elegant nor pedantic.

The field indicated by the title is exceedingly well covered, very little is omitted, and the reviewer has discovered only one misstatement: that hoary

and generally credited error, that in trigeminal neuralgia the nerve trunks are tender at their points of exit. Not only are the methods of examining the motor and sensory systems, the reflexes, the gait, the special senses, etc., clearly and completely given, but electrical examination and examination of the spinal fluid are included, as well as some very practical hints on simulation. Furthermore, the appendix of twenty-one small pages covers surprisingly well the Binet-Simon tests, aphasia, diplopia and the vestibular tests of Bárány, the last described and explained with unusual clearness and simplicity. Indeed, the thoroughness of the work, considering its size, is surprising. A few unimportant omissions may be noted. The pilomotor reflex is not mentioned nor that the cremaster reflex often is best elicited by sharp pressure on the inner surface of the thigh above the condyle. While lateral deviation of the umbilicus in tensing of the abdominal muscles is mentioned, the much more important Beevor's sign (upward movement of the umbilicus in transcord lesion at about the tenth dorsal segment) is omitted, as is the rectus jerk which may be quite informative. In the category of signs distinguishing functional from organic paralysis, Hoover's sign is not mentioned. Under "Movements of the Head" the characteristic attitude and movements of spasmodic torticollis might well be noted. Probably every experienced neurologist will fail to find a few of the tests or tricks he has learned to use, but he will find a good many he has overlooked or fails to use. Perhaps in future editions the author will take a little more space to indicate the significance of a few more of the signs and symptoms.

This little book will fit into a coat pocket or a hip pocket. It is good reading for a motor car, a street car or a railway car, and if a few thousand practitioners and a few hundred young neurologists of this country would commit its contents to memory, the American neurological level would rise like the Wissahickon in a spring freshet.

Fifty cents each will be paid for the April and May, 1919, issues of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.